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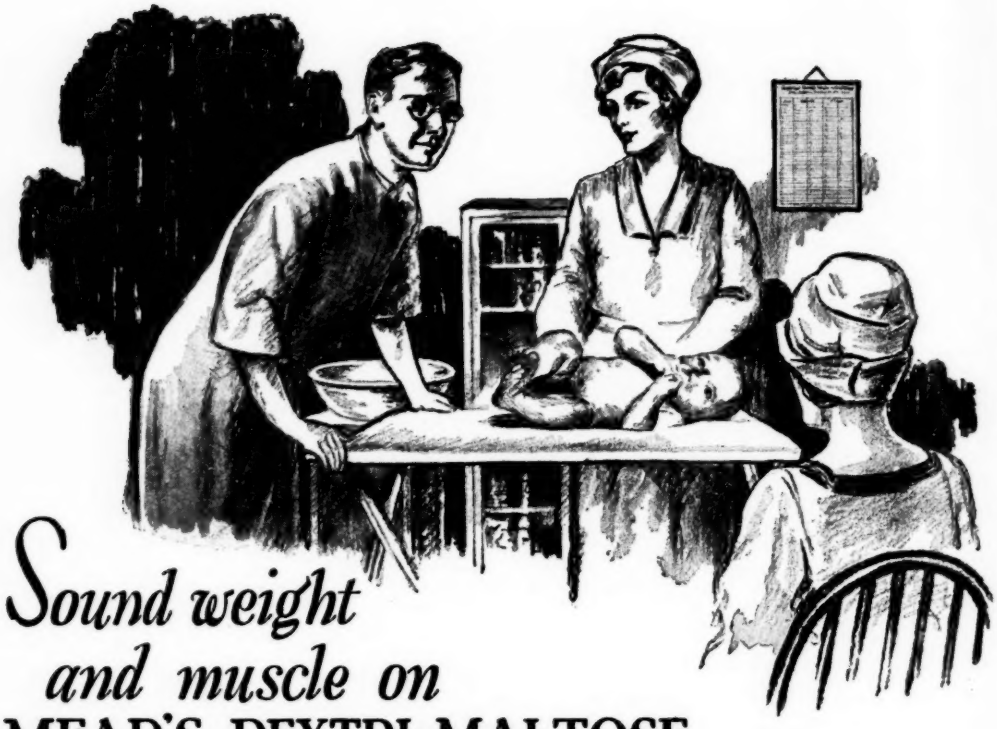

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MINNESOTA MEDICINE

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society.

VOL. XII AUGUST, 1929 No. 8

THE MORE COMMON INTESTINAL PARASITES OF THE NORTHWEST*

PHILIP W. BROWN, M.D.
THE MAYO CLINIC
Rochester, Minnesota

A CONSIDERATION of intestinal parasites which are most frequently encountered throughout the Northwest involves that of the various types of amebæ, flagellates and the beef and fish tapeworms. Round worms are infrequently seen in this section and the pork tapeworm (*Taenia solium*) is almost extinct here. Pin worms (*Oxyuris vermicularis*) must be fairly common, especially in children living under unsanitary conditions. Trichina, like the pork tapeworm, occurs infrequently; this is due to vigilance in teaching people not to eat raw or smoked pork. Only one ciliate need be mentioned, and that more for completeness, namely, the *Balantidium coli*. This parasite is common in hogs. Only seven cases of infestation with this parasite have been seen in the clinic.

There has been increasing appreciation of the distribution of *Endameba histolytica*, but with this has come even greater confusion as to the significance of non-pathogenic amebæ and flagellates. The parasites which have given rise to this confusion are *Endameba coli, Endolimax nana, Endolimax williamsi, Trichomonas hominis* and *Chilomastix mesnili*. Another flagellate which seems to furnish more tenable grounds for differences of opinion as to its significance is *Giardia lamblia*.

As a basis for study, I reviewed the cases seen in The Mayo Clinic during the first six months of 1928, in which any type of ameba or Giardia was identified. There were 184 cases, of which. in eighteen cases, infestation was with *Endameba histolytica*, in ninety-eight with *Endameba coli*, in thirty-nine with *Endolimax nana*, in fifteen with both *Endameba coli* and *Endolimax nana*, and in fourteen with Giardia. This is no indication of the incidence of the various parasites. The group of 184 cases is selected inasmuch as examinations of stools or suitable studies were undertaken in an investigation of various abdominal complaints.

There were thirty-three patients whose complaint on admission was of steady diarrhea, and seventy-three patients whose complaint, or one of whose complaints, was of intermittent diarrhea. Of these 106 cases, *Endameba histolytica* was found in only eighteen and in two of these there was chronic ulcerative colitis. I believe that this incidence of eighteen cases of infestation with *Endameba histolytica* in 106 cases of diarrhea, or only 17 per cent of a selected group of cases, is deserving of careful consideration. Although it is of utmost importance to recognize the sixteen cases of infestation with *Endameba histolytica*, the explanation of the diarrhea in the remaining ninety cases must be searched for along other lines. In some of them the diarrhea proved to be due to chronic ulcerative colitis, in some, to achlorhydria, in others to irritable bowel in nervous persons, and in a few there seemed to be a definite relationship to dietary deficiency.

Endameba coli, Endolimax nana, Trichomonas hominis or *Chilomastix mesnili* should not be accepted as explanations of the dysfunction of the bowel. There occur cases in which repeated examinations of stools disclose many or all of these parasites, which may lead one to assume that such heavy infestation must be a factor in the symptoms. There is the occasional case, in which one or more of these parasites

*From the Division of Medicine, Mayo Clinic, Rochester, Minn. Read before the Minnesota State Medical Association, St. Paul, May 13 to 15, 1929.

is the only abnormal element found by laboratory examination, and in which antiamebic treatment results in definite clinical improvement. I consider such a case as signifying either that *Endameba histolytica* was present and was not identified or that the emetin and organic arsenicals acted in a purely non-specific manner, such as is well known in the use of arsphenamine in the treatment of tuberculids.

In a small group of twenty cases in which *Endameba coli, Endolimax nana,* or both, were the only parasites found, antiamebic treatment was administered. Although it was not held that these amebæ explained the intestinal symptoms, yet treatment was instituted on the empiric basis that *Endamoeba histolytica* might be present but not identified. Of the twenty cases there seemed to be benefit derived from the antiamebic treatment in eight, whereas little, if any, response occurred in twelve. In the eight, it is uncertain just how definite a part was played by the specific treatment, as a suitable dietary regimen, dilute hydrochloric acid, and so forth, were employed. Opportunity was not afforded to make sufficient examinations of stools after treatment to determine whether the *Endameba coli* or *Endolimax nana* was eradicated; yet, even in the eight cases in which results were favorable, the response to treatment was in no wise as striking as it is in infection with *Endameba histolytica.*

The presence of these organisms in cases of normal or constipated action of the bowel can be even more fully disregarded. They were found in such cases merely as the result of the examination of a stool for occult blood as a routine, because of the history of passing segments of tapeworm, because of the patient's request, and so forth. *Endameba histolytica,* which is found in cases of normal or constipated action of the bowel, is of significance only from the standpoint of carriers, or the ever possible chance that dysentery might follow an acute illness or a surgical procedure.

I have avoided consideration of *Giardia lamblia,* as there seem to be reasonable clinical grounds for holding that this parasite, which infests the upper part of the small bowel, may be a factor in diarrhea. Experimentally or statistically there is little, if any, evidence for its pathogenicity. Furthermore, it is most rebellious to eradication and often it is hard to be sure whether it is or is not causing trouble. Fourteen cases of infestation with *Giardia lambia* were recognized during this period of six months; seven of the patients who were affected complained of spells of diarrhea and seven had normal or constipated action of the bowel. Treatment may be indicated in cases in which there are spells of diarrhea, but in the others the small risk of treatment is even greater than any good results that might be obtained.

The references that are made from time to time to the effect that *Endameba histolytica, Giardia lamblia,* and the other amebæ and flagellates mentioned here may be one of the etiologic factors in various forms of dermatosis, reflex abdominal complaints, arthritis, iritis, neurosis, and so forth, seem to me incorrect. Furthermore, valuable time may be wasted in employing treatment for infestation with these parasites. As a general working rule, even the one parasite which we all accept as pathogenic, *Endameba histolytica,* can be considered as an explanation of the patient's complaint only in the presence of diarrhea or formation of abscesses. I urge eradication of *Endameba histolytica* in any case in which it is found, but much explanation and trouble will be avoided by realizing that treatment seldom will be of subjective benefit if the patient's stools are normal or constipated. It is obvious that a neurotic person may be suffering from diarrhea, and although *Endameba histolytica* is found and treatment for it cures the diarrhea, one does not expect to cure the neurosis. But much less often will one influence, except unfavorably, a neurotic patient whose stools are normal or constipated by presenting to him, other than along very casual lines, the problem of eradication of amebæ. I emphasize this fact because the question of endamebiasis and neurosis has been the cause of more difficulty than even the question of the relationship of the ameba to arthritis; not that there is doubt concerning it, but the number of patients who consult the clinic with this problem seems to be increasing.

During 1928, thirty-five cases of *Endameba histolytica* were identified. The residence of these patients is always of some interest, and I found that the distribution was scattered throughout various states. Although this is of some interest, yet it is difficult, if not impossible, to give any idea of the incidence of endamebiasis among patients seen at the clinic. The residence

by states would have to be computed and even then many patients give an address at the time of examination which has not been their residence when the infection was contracted. Of course the incidence of acute amebic colitis in the north temperate zone is not as high as that seen in the tropics, and in previous reports[1, 2] the larger proportion of the milder cases of intermittent diarrhea seldom with gross rectal ulceration has been noted among patients from regions other than the tropics.

In this small group of thirty-five patients, three had chronic ulcerative colitis, and although antiamebic treatment was administered, specific treatment for the ulcerative colitis was more important. *Endameba histolytica* occurs in less than 10 per cent of the cases of ulcerative colitis and is a coincidental feature, which may or may not be instrumental in producing some of the symptoms. The recognition of this fact is necessary in order to obtain a favorable result in the treatment of such cases. Each disease must be recognized in order to effect a cure, as neither disease produces, or results in, the other.

Six of the remaining thirty-two patients had normal or constipated stools and were considered to be carriers. Ten patients were suffering with steady diarrhea and sixteen complained of spells of diarrhea with more or less constant "bowel consciousness." Proctoscopic examination revealed typical amebic colitis in ten cases. The value of taking smears for examination directly from the bowel must not be overlooked. In two of the ten cases, three examinations of stools had revealed only *Endameba coli* until smears from the rectal ulcers were examined and disclosed *Endameba histolytica.* The barium filling enema of the colon is of little value except to aid in ruling out any other pathologic processes. The only roentgenographic disclosures in uncomplicated amebic colitis are those of spasm, particularly of the ileocecal coil, and these are not of great significance.

In the group of thirty-five cases, there occurred four cases of amebic abscess of the liver. In one, aspiration was not done but the response to antiamebic treatment was so striking and the case seemed so clearly to follow the course of other cases of amebic abscesses that there was little doubt of the diagnosis. In the other cases, aspiration was done in two and drainage in the third. *Endameba histolytica* was identified in the pus in each case. In the preceding seven years, only six cases of amebic abscess of the liver had been seen and the fact that four were seen in one year is striking. The incidence of hepatic involvement in amebiasis is low in this region; probably in only a fraction of 1 per cent of all cases with amebæ does this complication develop.

For the last two years, the method of treatment employed at the clinic has evolved from an evaluation of the various compounds of ipecac or emetin and the organic arsenical preparations. Yatren (or anayodin) also has been employed in some instances, occasionally with excellent results, but more often the results have been discouraging. The high regard for the efficiency of ipecac and emetin continues and the response in a case of acute amebic colitis to the administration of 0.20 or 0.24 gm. (3 or 4 grains) of emetin hydrochloride is astonishing. Ipecac is unquestionably valuable but it also is hard on the patient and necessitates hospitalization. In the use of emetin hydrochloride these objections are avoided, but a serious objection to its use lies in the failure to effect eradication of the parasite in more than half of the cases. Use of arsphenamine was found to be successful in eradicating the amebæ, but the time and expense involved in a course of injections was an undesirable feature. The introduction of organic arsenical preparations that could be given orally has been an advance in the management of cases of infestation by amebæ. Stovarsol and acetarsone are essentially the same preparation. I have preferred to use a third preparation, treparsol. The therapeutic efficiency of the latter is not greater than stovarsol, but, as it is eliminated promptly, the risk of reactions is decreased. Treparsol alone is very efficient but the subjective response to treatment is enhanced by combining administration of emetin hydrochloride hypodermically, and treparsol orally. The patient receives 0.043 to 0.065 gm. (⅔ to 1 grain) of emetin hydrochloride twice daily for three days and this is repeated after a week's interval. At the same time, 0.25 gm. of treparsol is given with each meal for four days. The tablet should be chewed to facilitate more complete dispersion of the drug. The administration of treparsol is repeated for two or three more such courses, and eight to ten days are allowed to elapse between courses. This regimen has

seemed to effect cure in about 90 per cent of cases during the last two years. There is relatively little risk involved in using such a dosage of emetin hydrochloride, but there is an incidence of toxic erythema of 3 to 4 per cent from the arsenic. For this reason the patient should be seen daily during the first course, and at the least sign of reaction in the skin, administration of the drug should be stopped and it should not be used again. With such control, the danger of a more severe reaction, such as dermatitis exfoliativa, or even of death, is unlikely to occur. The possibility of peripheral neuritis being caused by either emetin or arsenic should be borne in mind, and if the patient already has had more or less recent treatment, yatren is a valuable second line of defense. The numerous other remedies, such as massive doses of bismuth, auremetin, bismuth-emetin-iodide, irrigations with kerosene, and so forth, must be resorted to occasionally, but persistence in carrying out a complete course of treatment as suggested obviates the need for the use of most other measures.

The intestinal worms which are more commonly met with in Minnesota and the neighboring states will be considered briefly: the cases of infestation with beef tapeworms (*Taenia saginata*) and fish tapeworms (*Diphyllobothrium latum*) vastly outnumber other varieties of infestation. The pork tapeworm has been referred to infrequently during recent years, and in the records of The Mayo Clinic there is not a single case of infestation with this parasite. As a result of a great deal of propaganda by parasitologists and public health officials concerning the proper cooking of pork, and the discovery of methods of killing trichinæ by freezing, the instances of parasitic diseases of man obtained from swine have been decreased.

The round worm (*Ascaris lumbricoides*) seems to thrive better in warmer climates and also bears a definite relationship to sanitation. In the Orient it is extremely common and various observers have noted that as high as 60 to 70 per cent of the population are infested. Ordinarily, the worm may cause few symptoms, although there seems to be present, in the infested patient, a certain degree of malnutrition and some nervous phenomena. These manifestations certainly could be explained partially on the basis of the patients' general habits of living. Reference also is made to toxic phenomena, such as headaches; they are attributed to the absorption of products derived from the worm, particularly from a dead worm which has not been passed out of the body. The wandering characteristics of the worm may produce serious mechanical obstruction by involvement of the common bile-duct, or of the larynx, producing fatal asphyxia. Another type of obstruction may result from a mass of dead worms that block the bowel. This has occurred when an anthelmintic has been used and has not been followed by a cathartic.

At present, there is not any entirely satisfactory method of eradicating round worms. For adults I prefer first to try 2 c.c. oil of Chenopodium in capsules given in 1 c.c. dose an hour apart; this may be given in the morning, with or without a light breakfast. Toxic symptoms such as nausea, vomiting, and signs of central nervous system irritation may result from absorption of the drug. This occurs much less frequently if the liver is well stored with glycogen. Also advanced cardio-renal disorders are contra-indications to the use of the drug. Two hours later, 30 c.c. of magnesium sulphate is administered. The stools are collected and carefully examined; usually only one or two worms are present. If this regimen fails, it may be repeated eight to ten days later, or thorough catharsis may be given in the evening, and the following morning 0.30 gm. (5 grains) of santonin followed by 0.065 to 0.12 gm. (1 to 2 grains) of calomel. The santonin is given on two successive days and repeated in a week if the stools still show the presence of worms or eggs. Occasionally a patient may suffer from yellow vision or vertigo and this should be explained to him, although it is not looked on as a serious complication when used in this dosage.

For years in The Mayo Clinic, from ten to fifteen patients infested with the beef tapeworm have been seen each year. It is surprising to learn of the number of persons who have a fondness for raw beef; not only the butcher who takes a bit from time to time, but also those who enjoy such delicacies as "cannibal sandwiches," which are simply raw ground beef with the addition of various condiments. As a result there continue to be cases of infestation with this worm. Magath and I were impressed by the almost total absence of any symptoms that could be attributed to the worm; it is only after the

patients become aware of its presence that almost any kind of symptom might be elicited.

Of even greater significance than the beef tapeworm is the fish tapeworm. At present there are not more than 200 cases of such infestation recorded from North America. It is well known that Dr. Nicherson of Ely, Minnesota, in 1906, reported the first case in this country in a native-born subject. The preliminary studies of Magath, in 1925, and his further report in 1927, emphasize the necessity of making every effort to prevent the spread of the infection. The identification of the larvæ of this worm in pike, pickerel, perch and trout shows how widely fish have been infected in certain lakes and the urgent need of instituting measures to prevent infection of fish in other lakes. Magath urges that sewage shall not be emptied into lakes or streams and that in an endemic center such as Ely an intensive campaign must be undertaken to teach people to cook fish well before eating it. Furthermore, any persons harboring the worm should be required to take treatment; the treatment should be verified by negative stools. The symptoms from the fish tapeworm are usually not more significant than those from the beef tapeworm. However, because of the severe anemia which sometimes accompanies the presence of the fish tapeworm, it is deserving of greater consideration.

Ehrstrom has made a fairly comprehensive study of this problem and he believes that anemia develops in only one of 5,000 to 10,000 cases. His studies were carried out in Finland; he estimated that there are from 500,000 to 750,000 carriers of this worm in Finland. In the small number of cases studied at The Mayo Clinic, none was associated with severe anemia.

The treatment, which has been previously described, is briefly as follows: The patient should not have luncheon or supper the day preceding treatment, although black coffee, tea or water may be taken freely. At 6:00 p. m. and again at 6:00 a. m., 15 to 30 gm. of magnesium sulphate is administered. Breakfast is not taken. At 7:00 a. m. and again at 8:00 a. m., 30 c.c. of emulsion of aspidium is administered. Two hours later, 30 gm. of magnesium sulphate is given and followed in another two hours by a large soapsuds enema. All stools, including the enema returns, are saved and carefully searched for the head. The enema is extremely important, for often when the worm is dislodged the head breaks off and passes into the colon, where it will remain unless swept out by a large enema. It is also important to use a saline purgative instead of castor oil, as aspidium is highly toxic and soluble in oil.

This procedure may seem unduly laborious but the method may be expected to yield successful results if each step is rigidly and precisely carried out. Many of the patients treated at The Mayo Clinic had been treated repeatedly, most of them four or five times, and one patient as many as eight times.

In trichinosis, treatment is of little value unless the disease is diagnosed at the onset of diarrhea, which is the time when the larvæ are maturing. During this period there is usually diarrhea and sometimes nausea and vomiting. A diagnosis may be made by the identification of the larvæ in the stools and any small epidemic of diarrhea occurring in a group of persons who have been eating the same food should lead to a careful search for the Trichina. Treatment at the onset is chiefly that of brisk purgation to sweep out the worms before the female has burrowed into the intestinal walls and deposited ova in the lymphatics. Thymol is recommended and should be administered. After invasion of the muscles, treatment is not of any definite value.

Treatment for pin worms is not complicated but must be carefully followed out. Since the greatest difficulty comes in preventing the subject from reinfecting himself, the most important step is to keep the hands from the anus. It is advisable for adults to wear canvas gloves at night and children should have their hands tied in the sleeves of their nightgowns. Underwear, night clothes and bed linen must be changed daily. The oral administration of 10 gm. daily of bismuth subcarbonate is advised by Leifer, and Chopra and Chandler suggest carbon tetrachloride in 3 c.c. dose, followed in two hours by 30 gm. of magnesium sulphate. This may be repeated in four to five days. A warm saline enema at bedtime, or 1 per cent thymol ointment applied locally, will help allay the irritation and permit sleep. Usually a week to ten days of this regimen will suffice to produce a cure.

SUMMARY

The significance of *Endameba coli, Endolimax nana, Trichomonas hominis,* and *Chilomastix*

mesnili is considered. The failure of these parasites to respond to antiamebic treatment is noted. A satisfactory method for the treatment of endamebiasis is reviewed. Attention is called to the occasional occurrence of amebic liver abscess in patients in the north temperate zone. Round worms are considered from both the symptomatic and therapeutic standpoints. The frequent occurrence and significance of the fish tapeworm is re-emphasized and treatment for it and the beef tapeworm is given. The recognition and prompt treatment of trichinosis are considered. Treatment for pin worms is outlined and the importance of preventing reinfection is emphasized.

BIBLIOGRAPHY

1. Brown, P. W.: Treatment of endamebiasis. Ann. Int. Med., 1928, ii, 177-191.
2. Brown, P. W.: A clinical consideration of endamebiasis. U. S. Vet. Bur. Med. Bull., 1928, iv, 752-755.
3. Chopra, R. N., and Chandler, A. C.: Anthelmintics and their uses in medical and veterinary practice. Baltimore, Williams and Wilkins, 1928, pp. 135, 144, 145.
4. Ehrostrom, R.: Die absolute und relative Haufigkeit der Betriocephalusanämie. Ztschr. f. klin. Med., 1928, cvii, 463-464.
5. Magath, T. B.: Experimental studies on *Diphyllobothrium latum*. Proc. Staff Meetings Mayo Clinic, 1927, ii, 169-171.
6. Magath, T. B., and Brown, P. W.: Standardized method of treating tapeworm infestations in man in order to recover the head. Jour. Am. Med. Assn., 1927, lxxxviii, 1548-1549.
7. Leifer (quoted by Abt, I. A.): Pediatrics, Philadelphia, Saunders, 1926, viii, 1063.
8. Mills, R. G.: Parasites, chiefly metazoan, observed in 7,000 specimens of feces from Koreans with an attempt to interpret the findings. Am. Jour. Hyg., 1927, vii, 222-263.

KERASOL AND KERAPHEN NOT ACCEPTABLE FOR N. N. R.

The Council on Pharmacy and Chemistry reports that in the advertising of the Picker X-ray Corporation, acting as distributor for the Kerasol Chemical Co., New York, the term "Kerasol" is applied to tetraiodophenolphthalein sodium and to capsules containing the drug. The Council further reports that under the name "Keraphen," with "Soluble Kerasol" as a synonym, the Picker X-Ray Corporation (as distributor for the Kerasol Chemical Co.) markets a mixture of tetraiodophenolphthalein sodium and tartaric acid. To avoid the confusion that is caused by the application of different names to the same substance, the Council does not recognize proprietary names unless they are applied by the discoverer of an article or by the one who discovers its therapeutic use, or with his consent. When the marketing of a product becomes open to general competition, the Council attempts to coin a convenient title for it, adopts this as the N. N. R. name, and then accepts the marketed brands only if they are offered under the N. N. R. name or the descriptive chemical name. In the case of tetraiodophenolphthalein sodium the Council adopted the contracted name tetiothalein sodium and adopted tests and standards for the control of products admitted to New and Non-official Remedies. Since the Picker X-Ray Corporation and the Kerasol Chemical Co. are not the discoverers of tetraiodophenolphthalein and since the marketing of this chemical in the form of capsules or in admixture with tartaric acid is not the discovery of these firms, nor of such fundamental importance as to justify the application of special names, the Council declared Kerasol and Keraphen inadmissible to New and Non-official Remedies. (Jour. A. M. A., June 29, 1929, p. 2171.)

DIPHYLLOBOTHRIUM LATUM (FISH TAPEWORM) INFESTATION IN CHILDREN

REPORT OF TWO CASES

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and
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IN recent years it has been shown that fish in the lakes in certain regions in Minnesota have become infested with the larvæ of the broad fish tapeworm, *Diphyllobothrium latum.*[2]

Buffalo fish and carp from Minnesota lakes and rivers are for sale in the city markets. These fish are used by many individuals, most of whom are unacquainted with the possibility of parasitic infection. Among the Jewish people particularly, the way is open for widespread infestation with *Diphyllobothrium latum,* because of the method of preparation of a popular Jewish dish, Gefüllte Fish. Pilot[4] and Barron[1] state that it is a frequent practice among Jewish women to taste the minced raw fish used in the preparation of Gefullte Fish, to determine the proper seasoning. In the two cases comprising this report, both the children were Jews and both mothers remembered having given small bits of raw fish to the children to eat.

Barron[1] found only sixteen cases of fish tapeworm infestation reported, in which the patients were natives of North America. To this series he adds three, which with the cases here reported brings the total to twenty-one. Of this number thirteen cases, or 61.9 per cent, occurred in Jews. This percentage may be slightly higher, since in two of the series the nationality was unknown.

The following cases are reported for the purpose of calling attention to the possibility of *Diphyllobothrium latum* infestation among children; also to add to the literature two more cases of infestation in native-born Americans.

Case 1.—M. S., a Jewish boy, five years of age, was brought in for examination because of attacks of diarrhea and abdominal pain. Close questioning elicited the information that about two years previously the child had been accustomed to eat small pieces of raw fish. Pickerel, pike, buffalo, carp and other fish, native to Minnesota lakes, were used by this family and raw pieces had been given to the child by the mother. The history of diarrheal attacks and abdominal pain covered a period of about two years.

Findings on physical examination were normal. Urine and blood examinations were normal. The stool examination revealed enormous numbers of ovæ of *Diphyllobothrium latum.*

The boy was put in the hospital, where he received treatment with the Oleoresin of Aspidium. Four hours after the last dose of the drug, six feet of fish tapeworm were passed. The scolex or head was not secured. The following day, at home, another portion of worm was passed. This specimen was thrown away by the mother so it was not examined. The scolex was probably passed at that time, however, since the stool was examined one month later and found free of ovæ.

Three months after treatment, the mother reported there had been no recurrence of the attacks of diarrhea nor of abdominal pain.

Case 2.—M. M., a Jewish girl of seven, had been seen on several occasions over a period of about three years, because of abdominal pain. There had also been one attack of diarrhea during this time. On direct questioning, the mother stated that she used to give the child small pieces of raw fish to eat.

Except that the child was overweight, the physical findings were normal. Examination of the urine and blood gave normal findings. Examination of the stool showed ovæ of *Diphyllobothrium latum.*

Treatment similar to that used in Case 1 was followed by the passage of five feet of fish tapeworm. The scolex was attached to the worm passed.

This patient has not been seen since the treatment, so we do not know if attacks of abdominal pain have recurred.

DISCUSSION

Stool examinations in these cases were made according to the method described by Rivas and Fife.[2]

The treatment used was as follows: No food was permitted except crackers and milk, or toast and milk, for 24 to 36 hours before drug treatment was started. At 8 p. m., about 12 hours before the first dose of the vermifuge, 4 ounces of citrate of magnesia was given. At 8 a. m. the following day, 0.5 c.c. of Oleoresin of Aspidium was given in an enteric capsule. This amount was repeated at fifteen minute intervals

until four capsules were given. One hour after the last dose of the drug, 4 ounces of citrate of magnesia was again given. The mother was then directed to have the child pass the next stool into a pan containing warm water at about body heat to prevent the worm from breaking off as a result of muscular contraction incident to marked change in temperature on being passed from the bowel.

In addition to the above procedure, Magath and Brown[3] advocate giving a soapsuds enema in the evening if the scolex is not secured at the time the worm is passed.

Some difficulty may be encountered in treating children in getting the patient to swallow the large capsules necessary to hold the Aspidium. This difficulty can be avoided either by washing the capsules into the stomach by means of a stomach tube, or a liquid preparation of Aspidium recommended by Magath and Brown[3] may be used.

This preparation includes the following ingredients:

Oleoresin of Aspidium	6.0 Gr.
Powdered Acacia	8.0 Gr.
Water q. s. ad	60.0 Gr.

1 tsp. of the emulsion contains .5 c.c. of Aspidium.

BIBLIOGRAPHY

1. Barron, M.: Infestation with *Diphyllobothrium latum* (fish tapeworm) with special reference to native cases. Jour. Am. Med. Assn., Vol. 92, pp. 1587-93, May 14, 1929.
2. Essex, H. E.: Early development of *Diphyllobothrium latum* in northern Minnesota. Jour. Parasitology, Vol. 14, pp. 106-108, Dec., 1927.
3. Magath, T. B., and Brown, Phillip W.: Standardized method in treating tapeworm infestation in man to recover the head. Jour. A. M. A., Vol. 88, pp. 1548-49, May 14, 1927.
4. Pilot, Isadore: *Diphyllobothrium latum* (fish tapeworm) infestation. Report of a case of native infestation in a child. Med. Clinics No. Amer., Vol. 12, pp. 211-213, July, 1928.
5. Rivas, Darnasso de, and Fife, Chas.: Intestinal parasitism in Philadelphia. Jour. A. M. A., Vol. 92, pp. 624-627, Feb. 17, 1929.

ALLERGY IN RHEUMATIC FEVER

Recently J. C. Small isolated a non-hemolytic streptococcus from the blood and various other tissues and fluids of patients with rheumatic fever. He considers this a specific organism and named it *Streptococcus cardioarthritidis*. Vaccines and serums prepared with this organism have received a limited amount of study in the treatment of acute rheumatic fever. Against the specific nature of this organism there is, however, a not inconsiderable mass of evidence. In addition to the evidence that questions the specificity of the rheumatic fever of the organism claimed to be the cause of rheumatic fever, work has been done which points to an allergic factor as being the cause of the disease. The problem of etiology in rheumatic fever is complicated; a study of treatment yields largely futility and despair. (Jour. A. M. A., June 22, 1929, p. 2102.)

TRYPARSAMIDE IN NEUROSYPHILIS

Tryparsamide is indicated in certain types of neurosyphilis and has been used in systemic syphilis by some; but its efficacy is probably much less than that of the arsphenamines. Tryparsamide has a toxic effect on the optic nerve and therefore should not be used in cases in which primary optic atrophy or neuroretinitis is recognized, either on ophthalmoscopic examination or suggested by the complaint of diminution of vision. During the course of treatment in an individual who has no pathologic changes in the optic disks, a careful ophthalmoscopic examination should be done before each injection in order to find the earliest possible neuritic damage. There are a variety of methods of treatment applicable to both systemic syphilis and neurosyphilis far superior to tryparsamide for the ordinary case and much less dangerous. (Jour. A. M. A., April 20, 1929, p. 1373.)

FACTORS OF SAFETY IN GALLBLADDER SURGERY*

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I AM going to condense what I have to say out of deference to the other speakers on the afternoon's program, for I realize we are going to be short of time. But let me say before I come to the subject of my paper that St. Paul has many distinctions. Its very name suggests apostolic successions. Medically and surgically it will hold an enduring place in the hearts of the members of the State Medical Association. For it was in this great city that the first cholecystectomy in America was performed—on September 24, 1885—43 years ago—by a past president of this organization, Dr. Justus Ohage, still in active practice.

Will the members rise in tribute and cheer him for so great an achievement.

In connection with Dr. A. S. Hamilton's interesting report, I would urge the members to support the Historical Committee in their efforts to record the epic story of Minnesota medicine —for within this State there have been and are now some of the greatest men the world has produced in the field of medicine. Minnesota can well feel proud of so great a contribution.

Sailing along the New England coast one man's craft strikes a rock and goes to the bottom; but where his boat sank, there men lift a danger signal, and henceforth, avoiding that rock, many fleets with their precious cargoes are saved. So with the pioneer medical men. With rare courage, they matched wit against emergency and proceeded over uncharted seas. They performed the first cholecystectomies, the first prostatectomies, the first thyroidectomies, as there are pioneering spirits today developing a new age in surgery—the physiological—under the courageous leadership of such men as Leriche, Cotte and Adson—and brought out in Dr. Adson's thrilling and brilliant demonstration this forenoon in the cure of thrombo-angiitis obliterans and Raynaud's disease.

These pioneers, what heroism and acumen they displayed! What a heritage they have left! As they chart new sea-ways, they leave warning signs and erect lighthouses to guide those who are to sail the perilous seas after them.

Factors of safety in biliary surgery lie:

1. In urging the patient to early operation, before complications set in.
2. In an accurate and complete diagnosis.
3. In a careful appraisement of the physical status of each prospective operative patient.
4. In careful and aseptic surgical technic.
5. In exercising sound judgment as to what procedure is best to pursue in each individual case—keeping well in mind while operating the patient's history and complaint.
6. In instituting not routine but individual post-operative care.
7. In having in mind at all times the best interest of the patient.

Early diagnosis and early operation in cholecystic disease is one of the largest factors of safety in the whole problem. If the gallbladder is infected it becomes a menace of large import, for it likewise exposes the liver, the intrahepatic ducts, the heart and the kidneys to serious damage. Furthermore, the chronic gallbladder, if not recognized and operated upon early, favors the formation of stones, which in many instances migrate to the common duct, causing obstruction of the duct with disastrous consequences, as for example, jaundice, blood dyscrasias, cirrhosis of the liver, acute pancreatitis, carcinoma. Deaver has put the situation of procrastination thus: "Every time I operate for calculous obstruction of the common duct, I feel that somebody has not been on the job, having failed to recognize the futility of medical treatment and neglected to have the gallbladder removed before the stone or stones migrated into the common duct."

A lessened mortality and fewer complications in recent years have resulted more from an appreciation of the necessity of a better preparation of the patient for operation than from an increase in technical skill, more through sound judgment than through speed, more through an accurate diagnosis than through a greater manual dexterity.

Why can Crile and Charles Mayo, in their

*Presented before the Minnesota State Medical Meeting, St. Paul, Minnesota, May 15, 1929.

toxic thyroid surgery, show a 1 per cent mortality today as against 10 per cent fifteen years ago? Their surgical skill is surely not responsible for the difference, for I am sure both had reached the summit long before then. Is not the reduction due rather to the pre-operative preparation as recommended by Plummer and the careful appraisement of each patient?

Why can the modern urological surgeon do a series of 500 prostatectomies in people in the sixty's and seventy's with only a 2 to 3 per cent mortality, as against the record of the equally or even more deft surgeon of ten years ago with an operative mortality of 10 to 15 per cent? Because, before the patient comes to the final operation in our day, he is decompressed, transfused, detoxicated, his blood urea reduced, his strength buttressed to the full against the ordeal by every means that medical science has at its command.

In the same careful and scientific manner is the patient prepared for surgery of the colon in obstructions due to malignancy or other causes, which is a prime factor in the reduction of operative mortality.

Modern medical training, apprenticeship, experience have taught the young surgeon of today wherein safety lies. He no more thinks of placing a patient on the operating table to undergo major surgery without a careful examination to ascertain his physical status, than he would allow himself to start out on an automobile trip with his family in the car without first checking up his steering wheel, tires, brakes and the amount of gasoline in his tank.

He makes more accurate diagnosis, that he may not only know the main ailment of his patient, but also the associated conditions that may, under the stress of his operation, come to the fore and defeat his purpose. This investigation will help him to balance the surgical insult imposed. He may be aided in the determination of his patient's condition, aside from what a careful history gives him, and the clinical findings, by the modern armamentaria which science and invention have placed at his command, if such are needed in the evaluation of his case. Electro-cardiograms, the roentgen ray, dyes, the cystoscope, the bronchoscope, the proctoscope, the Wassermann test, may all be used in the appraisement.

Trained as he is, he is less likely to mistake a cholecystitis for a root pain, a coronary for an upper abdomen. Nor is he apt to mistake a hydronephrosis or a stone in the pelvis of the kidney for a gallbladder condition, for his careful history-taking and a pyelogram, together with a Cole-Graham, will help him to differentiate—or discover both, if they be co-existent. He will not mistake an abdominal migraine for gallstones. He will not take a diabetic, arteriosclerotic hypertension patient and operate on him for cholecystic disease, without a careful pre-operative regime. He will not hurry an obese woman with co-existent myocardial degeneration to operation without a long pre-operative treatment by means of diet and rest.

Except in emergencies, he will not operate on a patient with even a suspected "cold." If he finds a post-operative oncoming pneumonia, he will add to his ordinary treatment the oxygen tent or introduce oxygen subcutaneously and see the distressed respiration and cyanosis disappear under it.

He will not operate on a deeply jaundiced patient without the three-day intravenous calcium chloride treatment—a la Walters.

He will never take a low-pressured, anemic, toxic, infected, dehydrated patient to the operating table until he has detoxicated, transfused and suffused the tissues with salines, glucose and sodium bicarbonate, as indicated.

He will select the type of anesthesia according to the patient's condition—regional, splanchnic, spinal, ether or gas.

He will heed Crile's warning as given in his chapter on "Operations on the bad-risk patient," when he says: "It would appear therefore that every patient should be subjected to the most searching scrutiny on the part of the surgeon to discover whether or not special supporting measures are required, and in case of even the slightest doubt those measures should be applied not after but in advance of the emergency."

He knows the factors of safety, too, in careful technic. He fortifies himself by an adequate incision—the lack of which has embarrassed one in many a situation, for it has proved the first cause of running into serious difficulties. He works cautiously near the cystic duct—for it is there where three-fourths of his troubles come, either through haste, lack of composure, poor orientation, or bungling. Here is where hemorrhage from the cystic artery may cause him, in

his excitement, to do irreparable damage to the common or hepatic duct—resulting not only in secondary operations with poor results, but even death.

He drains in every gallbladder case. He does a cholecystostomy rather than an -ectomy in the deeply jaundiced patient and in the acutely inflamed, edematous empyemic gallbladder. He does not delay operation in evident cases of threatened perforation, empyema, or gangrene.

The surgeon is a part of a great profession. He should never lend his art to ignoble purposes. He has great responsibilities, and should make it his duty to keep abreast of the times. His idealism should help him to realize, with Dr. Wilkie, that "grievous and irreparable harm may be done by his acts and his art thereby brought into disrepute. He should realize that it is not only the patient who is sacrificed; but that our cause is wounded in the house of our friends."

IRRADIATED ERGOSTEROL

Lest there still remain any misunderstanding, it should be recalled that the therapeutic virtues of cod liver oil are by no means to be identified with irradiated ergosterol; for the liver oil is rich in Vitamin A, which is in no way identical with the antirachitic properties of the irradiated ergosterol. The publicly announced statements that solutions of irradiated ergosterol represent the long desired "synthetic cod liver oil" are utterly misleading except as the Vitamin D component is concerned. Irradiated ergosterol cannot replace butter—a common source of Vitamin A—though it may supplement the valuable milk fat. When a highly potent substance such as irradiated ergosterol becomes readily available, it behooves us to consider carefully whether a danger of overdosage exists. While there appears to be a liberal range between a physiologically beneficent intake and a possibly injurious overdosage, there can no longer be much doubt that massive doses of irradiated ergosterol may result in considerable impairment of nutrition, loss of weight, pronounced hypercalcemia, and abnormal calcium deposits in certain tissues and organs. Investigators in the U. S. Public Health Service state that irradiated ergosterol is no doubt a useful drug and one endowed with great potency, but not without possible harm in the hands of the unsuspecting. Probably this is true also, the investigators add, of the haphazard consumption of food-stuffs that have been subjected to the action of ultraviolet rays. (Jour. A. M. A., June 15, 1929, p. 2023.)

CORAMINE-CIBA

The Council on Pharmacy and Chemistry publishes a preliminary report on Coramine-Ciba. The product is stated to be pyridine-carbonic acid diethylamide, proposed for use as a circulatory stimulus in cardiac failure, surgical shock, narcotic poisoning and respiratory failure. The product is marketed as Coramine Liquid, a 25 per cent solution for oral, sub-cutaneous, intramuscular and intravenous administration. The A. M. A. Chemical Laboratory confirmed in a general way the chemical claims. Eleven reprints were submitted by the Ciba Co., Inc., in support of the claims made; four of these referred to animal experiments, six to clinical results and one to both. In addition, the Council's referee examined a number of reports not submitted by the firm and reported the tenor of all to be favorable. The experiments on animals are not impressive, in view of the very large doses used to produce effects in animals. It is probable, however, that with a drug of this character observations on man would be most convincing, if they could be thoroughly controlled so as to exclude spontaneous changes. It is doubtful whether this is the case in the present instance, and while the clinical reports are generally favorable, they are not decisive. The Council voted to postpone action on Coramine-Ciba to await further experimental and clinical evidence which may establish its usefulness and to publish its report bringing out the experimental status of the product. (Jour. A. M. A., June 1, 1929, p. 1837.)

THE ACUTE PERFORATION OF THE DUODENAL ULCER*

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PERFORATION of a duodenal ulcer is a great catastrophe and is one of the most dramatic of the acute abdominal crises. I believe that this problem, of all those confronting the surgical staff in a large hospital today, is one of the most vital. Cases come in with considerable regularity. Contributions on this subject are frequent. Practically not a single month passes but one or two articles on this topic are published in the best journals. In the great mass of literature that has been published in the past, I have been able to find only one article[1] offering any laboratory research.

In this, the first section of my study, the objectives are to gather and to evaluate and correlate much of the recent data; also to review a series of some forty-six cases occurring at the Minneapolis General Hospital during the period of five years, from 1923 to 1928; and to determine what investigation either of a clinical or of a laboratory nature might be done to improve the mortality rate and the late results, none of which are very flattering. Not many can be very proud of their record in this field, as will be seen in the mortality chart.

It has been hoped that in working over this material some light might be thrown on the pathogenesis and the pathological physiology of peptic ulcer in general. More knowledge is needed in both these fields before the prevention and therapy of perforation can be improved.

My interest in this subject started in 1910, when I assisted Dr. Emil Beckman on the first case I had ever seen. Many will remember that in 1913, at the meeting of the American Medical Association in Minneapolis, Deaver read a paper on this topic which provoked a very lively discussion by Ochsner, Coffee, Mayo, Babcock, McKenzie and others. At many meetings since and especially at the American Medical Association meeting last summer, when Gibson read his paper, the battle still raged. It is evident that during the last twenty years, with all this agitation, there has not been the advance in this particular type of surgery that we have hoped for and have had every reason to expect.

In treating the subject I shall follow the conventional order of procedure, but will bring out only those things that seem of special interest under each heading.

HISTORY

To Mikulicz has been given the credit for the first suture of a perforated ulcer, in 1889. His patient did not live. Kriege was the first to suture an ulcer with recovery, in 1892.

ETIOLOGY

The real key to our success with peptic ulcer, and duodenal ulcer in particular—and hence perforation—lies in the discovery of the cause or causes. That this is a fertile field for study is only too well known.

Duodenal ulcer itself seems to be on the increase; naturally this means more perforations. In one clinic 2,261 duodenal ulcers were diagnosed by *x*-ray during 1928 and of these 803 came to operation. In this number there were thirteen acute perforations. Formerly 10 per cent, and now 12 per cent according to many authors (with Deaver putting it at 15 to 20 per cent), of duodenal ulcers perforate. Deaver goes on to say: "Hemorrhage and perforation are the chief and also the most frequent complications of the duodenal ulcer. Their frequency indicates the importance of the strict supervision of all ulcer patients. We believe that if patients were aware of the frequency of the inherent dangers, they would readily accept the conservative method of surgical intervention."

Duodenal ulcer is largely a disease of men, the ratio being 4 or 5 to 1—hence perforation is chiefly concerned with the male. In this series of forty-six cases all were men. Most of them were of middle age or beyond. W. J. Mayo, in attempting to explain the relative frequency of duodenal ulcer in the male, says: "In men the first portion of the duodenum passes upward. In women it is much more often transverse and

*From the Department of Surgery, University of Minnesota, Minneapolis General Hospital. Read before Minneapolis Surgical Society, March 7, 1929.

consequently in them it is alkalized by the bile and pancreatic juice."

The predisposing factors in perforation are not known. Many things seem to have a bearing, such as overloading the stomach, severe exertion, blows on the abdomen, barium meals with manipulations, passage of stomach tubes, acute alcoholism, eating the fruit of the papaya tree, etc. Many, however, perforate on an empty stomach, or even while the patient is asleep, or in bed on a Sippy regime.

One element I am convinced has a very definite relationship and that is an acute upper respiratory tract infection, an acute outbreak of oral sepsis, a mild bacteremia, or a toxemia of any sort. This relationship is evidenced by the fact that in our series several had had recent acute infections, some with a concomitant acute bronchitis or broncho-pneumonia. Again, we see the coexistence of fulminating infections of the appendix and an aggravation or actual perforation of a duodenal ulcer, following in the wake of an upper respiratory or other infection. In the future I would suggest that an attempt be made to obtain a blood culture on entrance of selected cases of ruptured ulcer.

Dental, tonsillar, and sinus infections, usually of a marked degree, were found in practically 100 per cent of the cases in this series. The percentage of concomitant infection in the appendix, gallbladder, and other abdominal areas was not so high. I believe that the records in this regard are incomplete or that some associated pathological process may have been overlooked in the need of prompt operation.

PATHOLOGY

It is now generally accepted that the majority of acute perforations occur in chronic ulcers. Moynihan presents a series of 218 ruptures where only six had no previous history of indigestion. In 117 deaths from perforated ulcer with autopsies in England, there were only twelve acute ulcers and in these twelve there were four that also had chronic ulcers and the latter was the one perforated in each instance. The results obtained in the various and ofttimes laborious researches on the pathogenesis of these chronic ulcers frequently seem discouraging. A good deal has been discovered, however, most of which is familiar to you. It is apparent that lesions similar to human duodenal ulcers can be produced by any one agent or a combination of a number of agents, such as vascular injury, bacterial injections, chemical or biologic toxins, trophic or glandular changes, heat or cold, and variations in the chemistry of the digestive juices.

Three comments concerning the cause of ulcer in general are of sufficient interest and ingenuity to be repeated here. Moynihan states: "Acute ulcer is a term used to describe a condition in which chaps, fissures, erosions, extensive surface destruction of the mucosa or even deeper layers are found. There is often clear evidence of an impediment to the vascular supply resulting in a defenceless area, of which the gastric juice makes haste to take advantage. The ulcers thus arising develop rapidly, often as terminal events, in consequence of a heavy infection or grave toxemia, and they probably heal rapidly. Acute ulcers are multiple. It is possible that a chronic ulcer begins in one of the acute ulcers that refuses to heal."

Brenner[3] in trying to elucidate the greater frequency of ulcer in the prepyloric region and the duodenum says: "The pendant portion of the stomach, which includes the distal part of the lesser curve, the pylorus and the first portion of the duodenum, has a blood supply that is considerably less than the stomach proper. This subvascularization is enchained by traction, when the stomach assumes a more vertical plane for a few hours after a meal. Moreover in this area the mucosa is intimately attached to the muscle layers with formation of rugæ. Thus no allowance is made for dilatation and traction and the area is predisposed to traction anemia. This ischemia lasts for two hours after meals and may be a potent predisposing factor for selective embolic infections, thromboses, or tissue digestion."

Einhorn is opposed to the belt in the prophylaxis and treatment of ulcer, apparently reasoning from the analogy of the frequency of ulcer in occupations where abdominal pressure is evident—cobblers, horse-shoers, etc.

Thus, in the description of the essential lesion of acute perforation, it is plain that there is a division into two great classes: (1) The acute perforation in the acute embolic ulcer; (2) the acute perforation in the chronic or already established ulcer. We are not concerned now with the perforations in the latter type.

The acute ulcer is usually a clear-cut "blow-

out" 2 to 3 mm. across, with minimum induration and reaction around about. The chronic ulcer presents four main types:[3]

1. The large ulcer with much scar tissue formation, encroaching upon the lumen of the duodenum and accompanied with more or less extensive extrinsic adhesions.

2. The medium-sized ulcer with moderate tissue infiltration, without narrowing of the lumen.

3. The small ulcer with a non-indurated or only slightly indurated base.

4. Multiple ulcers, including the so-called "kissing" type.

I believe it is important to remember that a non-calloused ulcer can be many years old. Most perforations occur in the last three groups. They are simple, single, round, slightly indurated 1 to 2 cm. in diameter, on the supra-papillary part of the duodenum (the proportion is 450 to 1) and nearly always in the first one and one-half inches of the anterior or superior surface.

The associated pathology consists of all grades of chemical and bacterial peritonitis, either local or general. Lesions of the appendix and gall-bladder are also frequently present. Broncho- and lobar pneumonia are found and at several European clinics emphysema of the skin, either local or general, is described in several instances.

The more commonly associated later complications consist in residual abscesses of all sorts and in all locations—especially subphrenic, hepatic, and pulmonary—also empyema and duodenal fistulæ.

SYMPTOMS

The symptoms of this condition are too familiar to require discussion in any great detail.

However, we should recognize that this is a complication of the chronic ulcer and hence often preventable. Management of the chronic peptic ulcer must be more strict, rather than "ambulatory" or dilatory as is frequently the case today, if our statistics are to improve. Surgery must be recommended more forcibly, at least in the cases that have had their nine medical cures. Greater publicity among the laity is necessary.

I believe we can now point out to you one other avenue of prevention. Certain ulcers, estimated at less than one-fourth, have prodromal signs and symptoms of perforation. There may have been very distinct exacerbations in severity of symptoms for days or weeks before—a warning that usually goes unheeded. Evidence of bleeding may precede perforation. Failure of the usual food relief sequence and the occurrence of night pain in patients formerly free from it, together with tenderness and rigidity in the epigastrium, are often premonitory signs. Excessive spasm, as noted in the *x*-ray, should put the observer on guard.

As regards the symptoms of a typical attack I will repeat an apt expression:[4] "The leading symptom is pain, the leading sign is rigidity, and the leading clue is a previous history of indigestion."

A word in regard to this question of previous history of dyspepsia. These people are often in such agony that no previous events can be made to seem important to them. You may have to reserve this part of the story until after the operation. Again, it is said: "These patients have often had indigestion so long they have ceased to remark upon it: it has been a part of their daily condition to which they submit uncomplainingly."

The terrific, agonizing pain of sudden onset, the unyielding rigidity often in a scaphoid abdomen, together with the wild-eyed anxious expression, costal breathing, cold sweat and "set" immovable posture, are too well known for further comment.

True surgical shock does not occur at the onset, as the pulse is not accelerated and the blood pressure is normal. This term is used too loosely and should be discarded. Collapse or prostration would be a better expression.

Briefly concerning this collapse, it may be said that with many patients it is the predominating sign from the onset to the time of their death in one to three days. They never apparently rally. It is a sort of "solar plexus" blow. Peritonitis, as we think of it, has not had time to develop. Several writers now are offering the suggestion that one great contributing factor is the absorption from the peritoneum which has been inundated with chemically active digestive juices. "There is a suggestion in some experiments now being conducted by Wangensteen[5] and some of his associates in the experimental laboratory of surgery at the University of Minnesota, that collapse that attends perforation of an ulcer into the free peritoneal space may have its ex-

planation in a peritoneal reflex. Symptoms dependent on a reflex mechanism for their causation are always subject to a wide range of variability, and the great variation in the degree of prostration exhibited by patients following perforation may find elucidation in such an explanation."

The rather fleeting supra-clavicular pain was only noted twice in this series. It is significant when present and is undoubtedly a phreno-spinal accessory reflex.

The obliteration of the liver dullness is more misleading than of value. It is difficult to elicit at best. Gibson says:[6] "In my experience it is of the greatest rarity." Spontaneous pneumoperitoneum can be demonstrated by the fluoroscopic screen or film with the patient lying on his side on the stretcher. Forcing this type of examination is hardly justified in the average case.

Nausea and vomiting are not always such a prominent feature as one would expect. Especially is this true when compared to acute pancreatitis and appendicitis. However, in some series it is common, being 40 per cent in the duodenal cases reported by McCreery[7] from the New York Hospital.

The findings gradually change as the peritonitis develops. A period of so-called "fatal improvement" may be observed but soon the typical picture of a distended "silent" abdomen with diffuse peritonitis is evident.

DIAGNOSIS

In the diagnosis of this condition it is often not a matter of trying to be so exact in the differentiation nor is it one of "watchful expectancy." It is sufficient to determine that a surgical emergency exists and to proceed to prepare for its treatment. Speed in bringing surgery to these people is the one great essential. As W. J. Mayo says: "It is better to be operated within twelve hours by a surgeon of little experience than by the most skillful surgeon in twenty-four hours." Cases where an acute perforation has not been found usually have some other condition requiring surgical treatment.

Briefly the direct diagnosis will depend upon the history of previous dyspepsia, a sudden severe epigastric pain followed in rapid succession by any or all the subjective and objective findings mentioned above.

The whole picture of this disease is so characteristic in the typical cases, that once a perforation is seen it will never be forgotten. Most ruptures are easily recognized in the receiving room. A borderline case may be confusing but they are unusual. Formerly mistaken diagnoses were reported but they are less and less frequent today. When Moynihan's list included forty-nine cases, the first incision had been made over the appendix in eighteen instances. E. H. Pool[8] in 1922 chronicled fifty-nine cases with an incorrect diagnosis in seven—six appendicitis and one cholecystitis. In only one instance in our series was any grave error made. In that case the appendix was removed and nothing more done.

In the differential diagnosis, the following would appear as of greatest value to be carried in mind: Appendicitis, perforated gastric ulcer, disease of the gallbladder or ducts, acute pancreatitis, acute intestinal obstruction, diaphragmatic pleurisy or pneumonia, vascular accidents such as coronary thrombosis or thrombosis in the mesenteric vessels, and ruptured ectopic pregnancy.

I have prepared a more detailed outline for differential diagnoses with regard to certain of these conditions, which is of value for review.

Acute Appendicitis.—The two diseases may coexist.

Similarity—

Pain: often sudden and in epigastrium or umbilical region and later on right side.

Tenderness: In right and often left fossa.

Dissimilarity—

History: may be the same. Not usually the duodenal ulcer type of early history.

Rigidity: not so intense and unyielding.

Agony: not so intolerable.

Respiration: not so strictly thoracic, as diaphragm is not so tightly held.

The relative tenderness and rigidity are different.

Acute Pancreatitis.—

Similarity—

The intensity of the agony at least equals and often exceeds.

Collapse is greater and more fulminating.

Dissimilarity—

History: often early history of gallbladder disease; often stones.

Pain in pancreatitis always worse in the epigastrium and it is there that tenderness, resistance, and distension are found.

Enlarged and edematous pancreas may be found.

Pulse: bad from the start. Often thin, poor quality, and rate may be disproportionate.

Vomiting: usually more prominent.

A peculiar cyanosis or lividity, particularly of the face, is frequent.

Pancreatitis occurs more often in corpulent people—often in women during early months of pregnancy.

General distension is rare.

The right side of the abdomen is not especially involved.

The hurried, jerky respiration due to fixation of the diaphragm is not seen.

Gallbladder and Ducts.—

Acute perforation may exactly simulate.

Careful previous history is the only possible aid in differentiation.

Acute Thoracic Disease.—

Similarity—

Possibly this simulates acute appendicitis more, but it can imitate perforated ulcer very closely. The fact that the explanation for the symptoms and signs lies above the diaphragm is something that the surgeon must never forget.

These points may aid in differentiation:

1. In chest conditions the initial temperature is often higher; in perforation it seldom is more than 102° in contrast to 103° to 105°.
2. Rapidity of respiration (forty or more) in chest conditions.
3. Disproportion between pulse and respiration.
   Chest—Pulse 100, and respiration 34 to 45.
   Abdomen—Pulse 120, and respiration 25.
4. In abdominal conditions there is never the same unchanging resistance of the abdominal wall and tenderness is more of the exquisite superficial type. The area affected is less and degree of involvement is less.
5. There is a comparative tranquillity of the lower chest where the chest organs are involved.

TREATMENT

It is in the treatment that the greatest divergence of opinion is found. Perhaps this is not possible to standardize owing to the nature of the disease. Various opinions and arguments will be presented in as impartial a manner as possible.

Certain ideas may be said to be fixed, and one of these is that there is a prophylactic side to the treatment of perforations. Relative to this point allow me to quote Moynihan at some length:[4]

"The duty thus laid on our profession is clear. We must take pains to become familiar with the signs and symptoms of chronic ulcer of the stomach and duodenum, so that this fatal catastrophe may be prevented. Perforation is a sign of neglect of some one—either the patient or doctor. It is not important that we should have equal knowledge of all diseases: it is important that all who are in general practice should have the clearest knowledge of the diseases which immediately and irrevocably assail life.

"Failure to recognize the emergencies that we are now considering means the loss of a man's life. Not less than 90 per cent of the perforations concern chronic ulcers.

"The position is serious, as a review of hospital reports from all parts of the country will show. It is our bounden duty to do what we can to improve it."

In the treatment of the acute rupture all agree that immediate surgery is the great deciding factor. Once the emergency has been determined a suitable opiate is administered, stomachal aspiration is performed if necessary, and a small amount of methylene blue may be given. Your patients may or may not be dehydrated, and it is also reasonable to presume that many of them have the disturbed ration of blood chlorids, urea and carbon dioxide combining power, practically always seen in pyloric and high duodenal obstruction, either of a transitory or chronic nature.

In the operative technic a combined local and ethylene anesthesia will be suitable for the majority. The individual surgeon's judgment must govern whether he will do a simple closure, cauterize the edges of the rent, excise, or do a pyloroplasty according to the Horsley, Finney, Mayo or Judd technic. Again, his judgment will enter into whether the closure will consist of one through-and-through stitch reinforced by a purse string, or interrupted stitches put in the way the tissues fold in the easiest, or on the Heineke-Mikulicz principle. Reinforcement of the closure with free or attached tags from the gastro-hepatic or gastro-colic omentum, or a piece from the suspensory ligament, must be considered. Any man's judgment in the above matters will be influenced by many factors, principally the time elapsed since perforation, the type, the size, and the position of the ulcer, also the interval since a meal, his opinion of the infectivity of the duodenal contents, the general condition of his patient, and the operating facilities available, together with the skill of his operating team.

Furthermore his judgment will be influenced by his own experience, his training, his keenness of observation and the data he may have at hand. Again, his decisions and actions may be determined by his knowledge of the experience of others. Therefore as regards the battle ground for and against a primary or secondary gastro-enterostomy I would like to go into more detail.

Braun in 1897 did the first primary gastro-enterostomy in a perforated ulcer. Moynihan claims the priority, having done his first in 1901.

He has continued to advise this operation since, while in this country Deaver has been its strongest advocate. The New York group led by Gibson oppose gastro-enterostomy; some say "never," and some "well, at times."

With your indulgence I will quote from several authors and then sum up some of the reasons given for and against.

Brenner[3] in twenty-seven cases had 60 per cent that required a second operation. He says: "Within the first twelve hours a simple closure will result in a cure in the vast majority of cases. The cardinal indication for gastro-enterostomy would be actual or organic obstruction." He reserves primary gastro-enterostomy for large calloused ulcers where closure produces a real stenosis and where there is already altered gastric physiology or function. A good practical rule he says is: "When in doubt—don't. It can be done later."

Gibson has a dozen articles on perforation in the more recent literature. In one essay he states that nineteen patients or 15.4 per cent of that series required a second operation and two had a second operation for perforation. It may be argued, he says, that if they had been more liberal with a prophylactic gastro-enterostomy they would have had fewer reoperations. His rejoinder to that is, that they might have had fewer living patients to operate upon. In another paper he admits he does not attempt to settle the vexatious question for or against gastro-enterostomy.

E. H. Pool[8] believes that 33 per cent will require a second operation.

Smith[9] in forty-one cases with simple closure enumerates four that had a second operation.

Cutter in reviewing several recent series concludes in favor of gastro-enterostomy.

D. Guthrie in his questionnaire had replies from 150 surgeons:

Twenty-four did gastro-enterostomy routinely (or pyloroplasty).

Sixty-four never.

Sixty-three varied procedure with circumstances.

Lewisohn[10] of Mt. Sinai has probably followed up his cases longer and with greater scrutiny than many. His study includes careful histories, physical examinations and *x*-ray series, in thirty-three cases operated between 1915 and 1925, and reported in the Annals of Surgery for May, 1928. He concludes: "First, a closure of an acute perforation of a gastro-duodenal ulcer (with or without gastro-enterostomy) failed to cure the patient in 39 per cent of the cases. Second, in those patients which were subjected to a partial or subtotal gastrectomy we often found another ulcer had been overlooked at the time of the primary operation. We have learned from our experience with resection that multiple ulcers occur in about 50 per cent of the cases." He goes on to state that possibly Finsterer, Von Haberer, Kreuter, Schwarz, Bruett, Paul and others may be on the right track when they urge and practice partial gastrectomy in the perforated duodenal ulcer.

Collinson,[11] Moynihan's right hand man, collected the results in fifty-three cases of his own and in those of others.

In twenty-six patients without gastro-enterostomy, seventeen, or 65 per cent, are free from symptoms. Nine, or 35 per cent, have recurrence of symptoms.

In twenty-seven patients with gastro-enterostomy, nineteen, or 70 per cent, are free from symptoms. Seven, or 26 per cent, have had recurrence.

One died of another cause.

Collinson modifies the use of gastro-enterostomy according to whether or not the surgeon is adept in performing the operation. However, to both those who are experienced and those who are not, he says, "When in doubt—don't."

To make this study complete on this seemingly moot point, I have prepared a list of arguments for and against gastro-enterostomy as they occur in the literature.

FOR GASTRO-ENTEROSTOMY

1. Perforation, with closure alone, does not cure the ulcer in a large number of cases.
2. Gastro-enterostomy does not affect the mortality in properly chosen cases.
3. The danger of spreading infection by gastro-enterostomy is of theoretical rather than practical importance.
4. While gastro-enterostomy is not always successful in relieving symptoms or preventing complications, there can no longer be any doubt about its specific effect.
5. Suturing always narrows the lumen and gastro-enterostomy safegards against second-

ary perforations and the effect of subsequent stenosis.

6. While subsequent jejunal ulcer is a possibility to be recognized, its incidence is extremely low.

7. Gastro-enterostomy relieves tension on the suture, purse string or whatever is used to close the ulcer.

8. The operation changes the chemistry of the stomach secretion.

9. Coexisting ulcers, if present, are favorably influenced.

10. The risk of hemorrhage from the bed of the perforated ulcer is minimized.

11. The administration of food and fluid can be begun earlier and with greater confidence.

12. All these factors make for a reduced primary mortality.

13. Stenbuck[15] reports eighty-eight cases from Mt. Sinai in all stages where a gastro-enterostomy was done in addition to closure. Four cases died or 14 per cent as compared to 31 per cent for the whole eighty-eight cases. Many so treated were advanced cases with peritonitis.

14. Moynihan states in the Practitioner for May, 1928: "But the wisest opinions seem to have drifted towards a recongnition of the need of some procedure to counteract and control stenosis in a large proportion of cases of duodenal rupture."

AGAINST GASTRO-ENTEROSTOMY

1. It is unnecessary—perforation curing the ulcer.

2. It adds to the mortality.

3. There is danger of spreading infection in peritoneal and retroperitoneal cavity.

4. It is not necessary in 100 per cent of the cases.

5. Reperforation, hemorrhage and stenosis are exceptional sequelæ.

6. There is danger of subsequent gastro-jejunal ulcer.

7. Deaver, himself, has had one case of gastro-enterostomy where a large hematoma formed in the transverse mesocolon, which required drainage.

8. A. J. Ochsner[12] spoke against a gastro-enterostomy by an unskilled operator—stating that he thought lavage (and now duodenal tube) would give the same amount of rest and quiet.

9. Brenner favors simple closure in the "soft" ulcer type (61.5 per cent in his series of fifteen cases). In the calloused type (39.5 per cent of his cases), he used gastro-enterostomy.

10. John Garlock[13] in reporting 100 cases from the New York Hospital states that 99 cases had simple closure at the primary operation. Ten also had gastro-enterostomy at the time of the first operation. In all, ten cases, including one pyloroplasty, required a gastro-enterostomy later.

11. A. O. Wilensky[14] says that in the group of cases where there is perforation in the base of an established ulcer, it is better to do a simple closure of the perforation during the emergency. One can always reopen the abdomen, should there be sufficient indication to do so, and then carry out the procedures which seem best at a time when conditions are not complicated by the dangers of an acute perforated lesion.

12. E. H. Pool[8] in reporting 59 cases says: "Our figures indicate that two out of three cases do well after closure alone: that only one in three after closure develop pyloric obstruction that demands operation."

In my own opinion I believe that the surgeon should keep an open mind, individualizing when the abdomen is open. To state dogmatically that all cases treated by simple closure will result in cure is as illogical as to insist on a primary gastro-enterostomy as a universal procedure.

In a calloused lesion—"the old battle scarred type," as I like to call them—with a large flabby obstructed stomach, you just know that a gastro-enterostomy is going to do the greatest amount of good. In an innocent looking rent, operated early, and every advantage being on your side, you can proceed as you might in operating a case at the time of election. Given a patient with more fulminating symptoms, you are not going to be so free to choose. Of this I feel positive: a primary gastro-enterostomy gives an ease, a smoothness, and a freedom from irritating disturbances, during the immediate post-operative convalescence, that can be a great comfort to your patient, his relatives and friends, and—last but not least—to yourself. These are the anxious days, when the big fight is going on, especially in those men with great prostration or collapse, spoken of earlier in the paper. Also during that interval you can count on the gastro-

enterostomy helping to make your plication hold and preventing further leaking. Again, to these people you can give fluids and other foods earlier and with greater confidence. The relief, following your program of enteroclysis, hypodermoclysis and intravenous therapy, is frequently gratifying.

The question of drainage comes up now in its logical sequence. Here, also, the surgeon's judgment will determine his conduct. To take something from Collinson, I would say, "When in doubt—do." Penrose drains do not produce the difficulties we used to see with the stiff tubes. A Penrose is of much value, however, in the cul-de-sac. Dr. Abbot used to put two or three Penrose drains inside each other. It's a good scheme. A drain should not be placed directly down to the closure area. A single Penrose through the operative wound may announce a reopening of the perforation which might go unnoticed and undiagnosed in a stormy convalescence of a tightly closed case. Reopening of the perforation I have seen occur in both the drained and undrained.

MORTALITY

The mortality varies greatly (6 to 60 per cent) in different clinics and from time to time in the same clinic. The chart shows the percentages from widely separated hospitals. The causes of death quite naturally group themselves into three classes: those that die within the first one to three days from the collapse so commonly called "shock"; those that die in the second period from the toxemia of peritonitis; and those that die weeks or months afterwards from residual infections or abscesses within the abdomen or thorax—sub-phrenic, pelvic, hepatic or pulmonary—or thoracic empyema.[15] The mortality table of our cases follows, with the postmortem findings.

EARLY AND LATE RESULTS

I have reserved the report of the results obtained in the review of this group for the second section of the paper. It is my hope to gather the histories, the records of physical examinations, and the *x*-ray data to show you at a later meeting. Possibly, also to bring before you some of the patients themselves. The tables of results from several clinics are appended.

CONCLUSIONS

1. A clinical study of a series of acute perforated duodenal ulcers suggests several research problems that might aid in increasing our knowledge of the pathogenesis of ulcer in general, and that might help in reducing the morbidity and mortality of perforation itself.
2. Perforation occurs largely in chronic ulcer and is thus preventable in a larger number of cases. Wider publicity is needed to make the diagnosis readily recognized in the average case.
3. Standardization of the treatment is difficult and possibly not desirable.
4. Causes of death are variable and fall into three classes: those dying during the first to third days, or early collapse period, the intermediate or peritonitis period, and, finally, the late, residual abscess group.
5. The variability in the reports of late results from the larger hospitals adds to the uncertainty regarding the efficiency of our present method of treatment.

I wish to thank the management of the hospital, the chiefs of the two surgical services, and the various associate surgeons for the privilege of reporting these cases.

BIBLIOGRAPHY

1. Stewart, G. D., and Barber, W. H.: Acute perforated ulcer of stomach and duodenum. Ann. Surg., Phila., 1922, lxxv, 349-355.
2. Balfour, D. C.: Report of surgical procedures of the stomach and duodenum for 1928. Proc. Staff. Meet. Mayo Clinic, Rochester, Minn., 1929, iv, 62-63.
3. Brenner, E. C.: Perforated ulcers of duodenum; study of 27 cases. Am. Jour. Surg., N. Y., 1927, iii, 381-385.
4. Moynihan, B.: Perforation of gastric and duodenal ulcer. Practitioner, Lond., 1928, cxx, 137-174.
5. Personal communication.
6. Gibson, C. L.: Acute perforation of stomach and duodenum. Jour. Am. Med. Assn., Chicago, 1928, xci, 1006-1008.
7. McCreery, J. A.: Acute perforated ulcer of stomach and duodenum. Ann. Surg., Phila., 1924, lxxix, 91-99.
8. Pool, E. H., and Dineen, P. A.: Late results of gastro-enterostomy for gastric and duodenal ulcers, including acute perforated ulcers. Ann. Surg., Phila., 1922, lxxvi, 457-466.
9. Smith, F. K.: Diagnosis and treatment of perforated duodenal ulcer. Brit. Med. Jour., 1921, ii, 1068.
10. Lewisohn, R.: Late results in perforated gastro-

Mortality Table with Post Mortem

Minneapolis General Hospital

Year	Type of Ulcer	Procedure	Post-operative Complications	Died	Post Mortem
1924	Old calloused ulcer	Polya—Time after perforation?	Duodenal fistula	23 days P-O	Diagnosis—generalized septicemia—drain leads to blind end of duodenum. Anastomosis O. K.
1924	Chronic ulcer	Posterior—Polya—Time after perforation?	Empyema infected operative area.	108 days P-O	Diagnosis—diffuse purulent peritonitis. Double empyema. Pus along suture. Anastomosis.
1924	Chronic ulcer	Closure only—10 hours after perforation.	Vomiting for 2 days. Pneumonia 4th day.	7th day P-O	Diagnosis—Pulmonary embolism. No post mortem.
1925	Chronic ulcer	Closure with G. E. 7 hours after perforation.	Possible pulmonary complication.	2nd day P-O	Sudden collapse. Death. No post mortem. 1—Peritonitis. 2—Interstitial pneumonia
1925	Chronic ulcer	Closure only 3½ hours after perforation.	Severe "shock." Cheyne-Stokes resp. in A. M.	1 day P-O	No post mortem. Diagnosis—general peritonitis. Surgical shock?
1926	Chronic ulcer	Closure. Post G. E. Time after perforation?	Vomiting blood next day.	52 days P-O	Perforation of gastro-jejunal ulcer. Peritonitis.
1926	Chronic ulcer	No operation.	Died 2 days after admitted.	2 days P-O	Chronic ulcer. Death from hemorrhage.
1926	Chronic ulcer	Closure with Post. G. E. 10 days after perforation.	Broncho-pneumonia 3 days post-operative.	13 days P-O	Diagnosis—Broncho-pneumonia. No post mortem.
1926	Chronic ulcer	Ulcer not operated. Appendix removed.	Stormy course. Simulated peritonitis and ileus.	8 days P-O	Post mortem—small amount of exudate in peritoneal cavity. Ulcer adherent to liver.
1926	Chronic ulcer	Operated 54 hours after perforation. Closure—Abdomen full of pus.	Did not rally.	2 days P-O	No post mortem.
1926	Chronic ulcer	Closure—Post. G. E. 6½ hours after perforation.	2nd day broncho-pneumonia. General peritonitis.	3 days P-O	Diagnosis—Peritonitis, broncho-pneumonia. No post mortem.
1926	Chronic ulcer	Operated 9 hours after perforation. Excision of diamond-shaped area with closure and omental graft.	O. K. to 9th day, then condition became poor.	10 days P-O	Post mortem. 1—Fibrino-purulent peritonitis. 2.—Resection wound open 3—"Kiss" ulcer found on posterior wall.

MORTALITY TABLE WITH POST MORTEM—*Continued*

Year	Type of Ulcer	Procedure	Post-operative Complications	Died	Post Mortem
1928	Chronic ulcer	Large perforation. Closure and omental tag. 3 days after perforation.	Stormy post-operative course. Supra-pubic drain put in 2nd P. O. day. Marked gastric retention. Evisceration.	3 days P-O	No post mortem. Diagnosis—General peritonitis.
1928	Chronic ulcer	Closure and detached omental tag. 24 hours after perforation.	2nd day broncho-pneumonia.	5 days P-O	Post mortem— 1.—Perf. duodenal ulcer. 2—"Kiss" ulcer. 3—General fibrino-peritonitis. 4—Broncho-pneumonia. 5.—Cloudy swelling.
1928	Chronic calloused ulcer	Closure with post G. E. omental tag. 24 hours after perforation.	26 days O. K. then sepsis began. All symptoms of pyemia. Stomach O. K. all through.	41 days P-O	Post mortem— 1—subphrenic abscess. 2—Pyemia with abscesses. 3—Purulent pleuritis. 4—Chronic ulcer. Duodenum-peritoneal surface showed no signs of injury.
1928	Chronic ulcer	Horsley pyloroplasty. 3 hours after perforation.	Purulent drainage from wound. Empyema drained at 2 different times	63 days P-O	Post mortem— 1—Pyopneumothorax. 2—Pulmonary atelectasis. 3—Careful search of stomach and intestines failed to show any ulcer or operative scar.

duodenal ulcers. Ann. Surg., Phila., 1928, lxxxvii, 855-860.

11. Collinson, H.: Some present-day problems in the surgery of gastric and duodenal ulcers. Jour. Am. Med. Assn., Chicago, 1914, lxiii, 1184-1190.
12. Deaver, J. B.: Posterior gastro-jejunostomy in acute perforative ulcer of the stomach and duodenum. Jour. Am. Med Assn., Chicago, 1913, lxi. 75-80.
13. Garlock, J. (Discussion: E. C. Brenner): Am. Jour. Surg., N. Y., 1927, iii, 385.
14. Wilensky, O. A. (Discussion: E. C. Brenner): Am. Jour. Surg., N. Y., 1927, iii, 385.
15. Stenbuck, J. B.: Causes of death following operations for perforated gastric and duodenal ulcers. Ann. Surg., Phila., 1927, lxxxv, 713-721.

DISCUSSION

DR. C. B. WRIGHT: I appreciate the opportunity to hear this careful and thorough paper and congratulate Dr. Olson on being able to report such a large series of cases. A medical man can contribute little to the diagnosis or treatment of perforations as this is a surgical field purely, and results will depend upon the early resort to surgery. My own impressions are that this is not nearly as common an accident as some of the statistics would lead us to believe. Surgeons like Moynihan and Gibson see many of these cases because they have difficult abdominal cases referred to them. I have asked before a number of medical societies how many cases each man had seen, and the reports indicated that men in general practice have seen only a rare case and many none at all. I have inquired among surgeons in regard to this matter and none of them over a long period of surgical work had operated on more than fifteen cases in private practice. Therefore, I think that the statistics one reads are somewhat misleading.

In my practice, I have personally come in contact with nine perforated ulcers. Six of these were perforated duodenal ulcers. Two were seen about sixteen hours after the onset and died following operation. Two recovered from the operation. According to reports one died three years later from hemorrhage, and another six years later of gastric tetany. Two others are living but have had recurrent attacks of ulcer trouble over periods of ten and twenty-two years. Three were perforated gastric ulcers. Two of the gastric cases were operated and remained well. The third refused operation and died of general peritonitis. These

cases usually are emergencies and call the nearest medical help. Unfortunately we are not all Moynihans and Gibsons. Men who see only a very rare case of perforation naturally do not at once think of this accident. More often the patient himself hesitates to jump on the operating table but begs for relief of his pain and a few hours to let the storm pass over. Morphine may be given and a period of comparative comfort may last until the onset of general peritonitis.

As regards ulcer in general the treatment of the patient is just as important as the treatment of the ulcer. Foci of infection, generally believed as the most common inciting cause, should be removed as soon as possible. Proper advice should be given as to diet, which should be balanced in every way with sufficient vitamin content, yet non-irritating. There should be proper periods of rest and sleep and attention paid to the nervous system. There is evidence to support the view of Hurst that certain individuals have a tendency to ulcer formation, the so-called "ulcer diathesis." Therefor the patients should be impressed with the fact that they are liable to have recurrence of the old ulcer or the development of new ones.

Finally, ulcers cannot be treated on the basis of statistics from large clinics. Each case is an individual problem and should be carefuly studied from every angle before the type of treatment is decided upon. All cases treated should be interviewed at regular intervals as long as possible after a so-called medical or surgical cure.

Dr. O. H. Wangensteen: Dr. Olson is very deserving of commendation for his thorough discussion of this subject. There were several points in Dr. Olson's paper that interested me. First, the mortality: The mortality in perforated duodenal ulcer is a measure of the delay that has intervened between perforation and operation. We count mortality in appendicitis in days, but in perforation of the stomach or duodenum, the mortality rises with each hour lost.

In a typical case, anyone can make a diagnosis with relative ease, but in an atypical case it may be difficult. A board-like abdomen following a sudden severe abdominal pain means perforation. The early pain is usually as severe as any that the patient may subsequently suffer. I have a plate here I would like to show. The patient is a male about fifty-one years of age. Four years ago, he was in the University Hospital and a diagnosis of neurosis was made. About two weeks ago, while at work in his occupation of "barbering," he suffered a severe attack of pain in his abdomen, which lasted just a short time. He went about his business and had no further trouble. Five days later, about three o'clock in the afternoon, he had another severe attack. He called his doctor. His abdomen was rigid, but no findings of a peritonitis developed. Five days after this acute trouble, he was sent to the University Hospital. We felt he had an acute perforation of a duodenal or gastric ulcer.

The abdomen, on admission, was moderately rigid throughout but more marked above. The pulse was not unduly rapid, and the man, on the whole, looked fairly well. No free gas could be percussed in the peritoneal cavity. Fluoroscopic examination also failed to show free gas in the abdomen. When a little barium was given by mouth, it was found that the stream shot out of the superior portion of the duodenum into this pocket shown here on the radiograph. In view of the localized and well walled-off perforation, we felt it safer to temporize and not risk contamination of the general peritoneal space. A gastroenterostomy was done for this man about six weeks later, at which time dense adhesions were found in the upper right quadrant.

Another patient, aged about 30, at present on the medical service at the hospital, is of interest in this connection. About two months before her admission to the hospital, she had acute pain in the abdomen. There was no history of ulcer. Free gas could be demonstrated in the peritoneal cavity. She is not ill now; pulse and temperature were normal, and there was no evidence of ulcer. The most likely occurrence here, I should think, would be a minute perforation. Why the gas has not been absorbed, of course remains unexplained. There are no gaseous cysts in the abdomen; there has been no severe trauma or antecedent operation to account for the presence of this free gas. Another remote possibility may be that air has been admitted to the peritoneal cavity through the vaginal canal by way of the uterus and Fallopian tubes.

These two cases illustrate the difficulty one can have in making a diagnosis in atypical cases.

The question of shock in perforation has been of interest to me, and we have investigated it a little from the experimental standpoint. I have never had the opportunity to see a patient directly after perforation. I have seen them two or three hours later. Then the pulse and blood pressure are usually normal. I don't know whether any observations have been made on blood pressure in such cases immediately following perforation.

It is my impression that the collapse that attends perforation is dependent upon a peritoneal reflex. I don't believe that the amount of hydrochloric acid that escapes into the peritoneal cavity, if injected directly into the blood stream, would cause shock. By injecting mustard oil into the peritoneal cavity of a rabbit, we have been able to demonstrate in the blood pressure tracings a fall in the blood pressure in some animals, and especially if the injection is made when the animal is allowed to awaken from the anesthetic. Anesthesia, of course, abolishes reflexes. A peritoneal reflex would be dependent on a nervous mechanism, and nervous reflexes are known not to be constant.

SACRAL ANESTHESIA*

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WITHIN the past two or three years sacral anesthesia has grown in popularity as the choice of anesthesia for operative work in the perineal region. To me it is the ideal type of anesthesia for rectal operations involving the hemorrhoidal and peri-anal region. It is simple of execution, as nearly devoid of danger as any other type of anesthesia and produces a degree of relaxation of the tissues which cannot be secured under general anesthesia. Over infiltration anesthesia it possesses the advantage of not distorting the field of operation and there is no danger of later hemorrhage such as may occur from an overlooked vessel where solutions containing adrenalin are used for infiltration anesthesia. It also possesses the advantage that the entire amount of anesthetic solution is injected with only one insertion of the needle, where several are needed to infiltrate. I believe that where alarming symptoms have occurred following sacral anesthesia it has been due in most cases to improper technic or the use of solution of improper amount or strength.

Various men use various solutions. The amounts seem to vary between 15 c.c. of a 3 per cent novocain solution to 90 c.c. of a 1 per cent solution. Adrenalin may or may not be added. There are certain definite disadvantages in using the large amounts of solution. It has been shown that 75 c.c. of solution injected through the sacral hiatus will sometimes extend as far as the third cervical vertebra, 30 c.c. to the ninth thoracic, 20 c.c. to the twelfth thoracic. With a 1 per cent solution at least forty to fifty c.c. are necessary for anesthesia and in many cases considerably more is needed. With the large amounts, very serious reactions consisting of a rapid thready pulse, respiratory embarrassment or collapse may occur. This is probably due to the effect on the nerves higher up along the trunk. Since only anesthesia of the perineum is required, theoretically at least, it is not necessary that the anesthetic solution extend above the lumbar region at the highest.

I had two patients who developed very alarming symptoms during the time I was using larger amounts of solution. Berry of Baltimore uses 20 c.c. of a 3 per cent solution without adrenalin and reports success in 86 per cent of his cases. While no doubt this is an ideal solution from the point of view of amount used, I consider any solution which will give only 86 per cent of successes as unsatisfactory. My own percentage of success was about the same as his with this amount and concentration of solution. After trying various solutions I now use 30 c.c. of a 2 per cent novocain solution to which .2 c.c. of 1-1000 adrenalin has been added. The amount of novocain solution is varied in individual cases between 20 and 40 c.c. This clinically produces results and I have had no severe reaction since using this solution. I believe the use of adrenalin is correct except perhaps in a few cases of cardio-vascular disease and hyperthyroidism. It prolongs anesthesia, slows up the absorption of the novocain and is a prophylactic against the type of reaction which most commonly occurs. Martin of Detroit has shown that the use of veronal also lessens the danger of toxic effects of the novocain and I have also added this to the method I employ.

Two types of reaction may occur with this type of anesthesia but as stated above I believe that there is no occasion for them to occur except perhaps in a very mild and transitory form, if the procedure is properly carried out. I will, however, describe them. The first type is by far the most common and is said to constitute 85 per cent of the cases which show reaction. In this type the patient complains of a sensation of weakness, is apprehensive and nauseated. The face is pale and perspiration stands out on the forehead. The eyes have a staring look and cerebration is retarded. The pulse rate falls along with the blood pressure. In severe cases vomiting or unconsciousness may occur. This type of reaction is controlled very quickly and effectively by the intramuscular injection of 1-1000 adrenalin in doses of 10 to 30 minims. Since adding adrenalin to the anesthetic solution I

*Read before Monthly Meeting of the Northwestern Hospital Staff, Minneapolis, May 8, 1929.

have not had a reaction severe enough to need any treatment of any kind.

The second type of reaction occurs very infrequently. This type shows restlessness, excitement, flushing of the face and acceleration of the pulse. Breathing is deep, the blood pressure raised and involuntary twitchings and unconsciousness may occur. This type is probably a true novocain intoxication. The treatment is simply to reassure the patient and keep him quiet. If the blood pressure is high amyl nitrite may be used.

I know of no complications which may develop later, except in one case an abscess was reported, but this no doubt was due to some error in asepsis.

Anesthesia is usually supposed to fail in about fifteen per cent of cases. I believe that anesthesia should be secured in approximately 100 per cent of cases where there is no anatomical anomaly which prevents the proper carrying out of the procedure. I believe that almost the only reasons for not securing anesthesia is that the needle is not in the sacral canal, enough solution is not used or sufficient time is not given. At least since our present plan we have had only one failure and I now believe that this was due to the fact that the needle was not in the canal. Unilateral anesthesia sometimes occurs. Lundy thinks this is due to the fact that the needle is not in the center of the canal. I think the most likely explanation is that the needle tip gets behind some bit of fascia or tissue and balloons out that side of the sacral canal but shunts the main part of the solution upward without filling out the canal proper. The chief evidence that the needle is in the caudal canal is the free flow of fluid from the syringe. A tingling down the thighs and legs sometimes occurs but not always, and later of course there is anesthesia of the perineum. Of course the aspiration of spinal fluid or blood is also evidence, but of an unwelcome sort.

Evidence that the needle is not in the canal is persistent resistance to the introduction of the solution, tumefaction from injection into the soft tissues over the sacrum and lack of anesthesia.

I claim no originality in the procedure employed for it is a combination of several ideas and for that reason a brief description is perhaps justified.

As a preliminary, five grains of veronal is given the night before and five grains one hour before the operation. A hypodermic of 1/6 to ¼ grain of morphine is given one half hour before operation. The patient is placed in a comfortable position, lying on the abdomen. If the sacrum is prominent he lies flat on the table; if not, a pillow is placed under the hips. The skin is cleansed with iodine and alcohol. The sacral hiatus is located in the usual way. With a fine needle the skin over this area is infiltrated with the procain solution and the needle is then inserted downwards and the tissues about the hiatus injected and about 1 c.c. injected into the hiatus. I believe this lessens the discomfort when the spinal puncture needle is later inserted. A spinal puncture needle is now inserted through the membrane covering the sacral hiatus. The distal part of the needle is depressed, so if possible the needle passes in through the center of the sacral canal and does not pierce the plexus of veins which lie against the bony walls of the canal. The needle tip is usually inserted about two inches, although in some cases anesthesia is secured to better advantage if the needle is not inserted so far. The stilet is now withdrawn from the needle and aspiration is done to see if either blood or spinal fluid is obtained. If blood is obtained the needle point is moved until no blood is withdrawn. If this is not done, intravenous injection of the solution may occur. If spinal fluid is obtained I believe it is best not to continue the procedure but to use another type of anesthesia. A number of men think that in this case the needle should simply be moved about until no spinal fluid is obtained and the injection continued. I know personally of one case in which this was done and when the solution was injected the patient promptly died. Obtaining spinal fluid is a rare occurrence. I have encountered it but once and in this case I discontinued the caudal procedure and had the patient given ethylene.

With the needle inserted in the canal and no spinal fluid or blood obtained the solution is injected. This is done slowly, the entire procedure taking usually from ten to fifteen minutes. I believe the slow injection is important. Certainly the number and severity of reactions may be reduced by slow injection. At first 2 c.c. of solution is injected. For some reason this small amount will give a slight reaction but as soon as

it has passed away the entire amount may be injected with no further trouble. The solution is injected at the rate of about 2 c.c. a minute until 20 c.c. have been injected. The anal region is then watched for any relaxation of the sphincter. If this begins we are sure that anesthesia will be secured. If, however, at the end of twenty minutes from the onset of injection there is not some evidence of this, another 10 c.c. of solution is injected. The needle is left in position so that if anesthesia is not secured at the end of forty-five minutes, more solution may be injected. This is not done until the needle's position is changed, for I believe if as much as 30 c.c. of solution is injected into the proper area anesthesia will be secured. Lack of anesthesia from this amount is sufficient evidence that the solution has not been properly placed. As before stated there has been only one case of lack of anesthesia, and no alarming reactions since following this routine.

In conclusion I wish to say that it is my opinion that with the present standardization of caudal anesthesia, it is the safest and most satisfactory anesthetic for the majority of cases requiring surgery of the perineal region.

BIBLIOGRAPHY

1. Berry, G. P.: Caudal epidural anesthesia in perineal surgery of the genito-urinary tract. Jour. Am. Med. Assn., 90:1018 (Mar. 31), 1928.
2. Labat, G.: Regional anesthesia. Saunders Company, 1928.
3. Lundy, J. S.: Method of producing block anesthesia of sacral nerve. Am. Jour. Surg., 4:262 (March), 1928.
4. Lundy, J. S., and McCusky, C. F.: Sacral anesthesia in operation on the rectum. Atlantic Med. Jour., 30:14 (Oct.), 1926.
5. Martin, E. G.: Local anesthesia agents; preventive and emergency treatment of toxicity. Trans. Am. Proc. Soc., 1928.

YEAST AND PUFFERY

The British Medical Journal has called attention to some "highly objectionable advertisements of a proprietary brand of yeast" that were appearing in American and Canadian periodicals. These advertisements are stated to have been of the testimonial type and purported to be signed by European or American medical men. While not mentioned by name, it seems quite obvious that the British Medical Journal referred to the blatant series of advertisements that the "Fleischmann's Yeast" concern has been running recently. As a result of the editorial comment, a well known London physician has written to the British Medical Journal that he was asked to write a testimonial extolling the virtues of yeast, this testimonial to appear with his name and photograph in magazines and newspapers, and was offered the sum of one hundred fifty pounds ($750). The physician did not accept the offer of the advertising agent. The following American physicians' names (and pictures) have been used by the Fleischmann people in their recent advertising campaign: "Dean H. H. Rusby, M.D., Professor of Physiology, College of Pharmacy, Columbia University." "Dr. George Parrish, well known Health Officer of Los Angeles." "Dr. Ira L. Hill, prominent New York physician and abdominal surgeon." (Jour. A. M. A., June 15, 1929, p. 2025.)

ELIXIR KACYAN McNEIL AND TABLETS KACYAN McNEIL NOT ACCEPTABLE FOR N. N. R.

The Council on Pharmacy and Chemistry reports that it decided not to admit potassium sulphocyanate to New and Non-official Remedies because the evidence for its therapeutic value is inconclusive. However, in view of reports in which reduction in pressure is clinically desirable, the Council holds that further consideration should be given the drug and voted to publish this report, stating the limitations of therapy with potassium sulphocyanate, namely: that the evidence for its value is far from conclusive; that in many patients the production of lowered pressure does more harm than good; and that its use is contraindicated in acute inflammation of all types, in nephritis and in marked renal insufficiency. The Council also reports that Elixir Kacyan McNeil and Tablets Kacyan McNeil are the proprietary names under which Robert McNeil, Philadelphia, markets an elixir and tablets of potassium sulphocyanate and that these are unacceptable for New and Non-official Remedies because the evidence for the value of potassium sulphocyanate is inconclusive and because the marketing of this drug under a proprietary name is contrary to the interests of rational therapy. (Jour. A. M. A., June 1, 1929, p. 1838.)

GENERAL TREATMENT OF TUBERCULOSIS*

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THE treatment of tuberculosis is such a many sided affair that in a short description of it many points of interest will necessarily be omitted.

The essential treatment of tuberculosis is very simple, so simple, in fact, that it requires a large amount of resourcefulness on the part of the physician to keep the patient on the "cure."

Without the time-tried remedies of rest, fresh air and proper food there is no cure for tuberculosis. The greatest of these remedies is rest. Without air and food life is impossible, but without rest the treatment of tuberculosis is futile.

The treatment of tuberculosis can best be applied in a sanatorium where the patient is subject to rules of a semi-military character, is under constant medical supervision, protected from well meaning but harmful advice-dispensing friends, surrounded by understanding associates, protected from many tempting lapses from the "cure" and where fresh air, good food and rest are not only constantly insisted upon but intelligently applied.

A patient with the usual, uncomplicated case of tuberculosis requires about the same amount of food that a healthy, active person does. The food problem is solved by the factors of sufficient quantity and variety. A variety, including fresh, green foods, assures the vitamin requirement.

The fresh air requirements of the patient can best be met by a well screened porch adjacent to a room into which the patient can be moved for bathing, dressing and for protection during bad weather. If such a porch is not to be had, the next best thing is a room with one or more large windows, the sashes of which have been removed, having screens and a canvas curtain at hand to keep out rain and snow.

Rest should be continuous as long as there is fever. If the patient has had a far advanced case of tuberculosis, the rest should continue for several months after the fever has subsided. Rest should be both mental and physical and should be as nearly perfect as possible.

A patient with fever should be supine in bed all of the time, not even being permitted to sit up in bed, let alone getting up for baths or to go to the toilet. A thorough understanding of tuberculosis and having witnessed the dire effect of too early a return to exercise are great aids in making one a safe judge as to the proper time for the patient to begin taking exercise.

Exercise should be limited to two or three minutes per day at first and gradually increased as the reaction of the patient warrants.

The thermometer should be used as a general guide in prescribing exercise. A rise in temperature of .5 of a degree F. or more that does not subside within about thirty minutes after quietly resting on a bed or couch, may be considered as an indication that the patient has had too much exercise. At first, even the act of sitting up in bed with the aid of a back-rest must be considered as "exercise." After the patient has been able to sit up in a chair for two or three hours he may begin to take walks, starting with a two or three minute walk. Walking, properly prescribed and taken, is one of the safest and best forms of exercise for the tuberculous patient.

Air-baths, natural sun and artificial-light baths, occupational therapy, suitable forms of amusement and all safe methods of helping to establish and maintain the "proper mental attitude" should be employed as conditions seem to require them.

Artificial pneumothorax, thoracoplasty, phrenicotomy and other forms of chest surgery are all life-saving measures, when used by men familiar with their limitations, and on properly selected patients.

All forms of tuberculous complications should be watched for and promptly treated, especially the more common ones such as pleurisy, empyema, laryngitis, enteritis, peritonitis, ischiorectal abscess, hemoptysis and tuberculosis of the glands.

The treatment of pleurisy and hemoptysis may be touched upon at this time as they are perhaps

*Read at the Tuberculosis Institute, held under the auspices of the Minnesota Public Health Association, Ah Gwah Ching, Minn., May 2, 1929.

the two complications that the general practitioner is most commonly called upon to take care of.

Simple dry pleurisy is best treated by the administration of salicylates and the application of mustard plasters or tincture of iodine. In cases of acute pleurisy, before the effusion appears, it may be necessary to administer morphine but at times strapping the chest with adhesive plaster will relieve the pain. Ordinarily, it is not advisable to tap fluid in tuberculous pleurisy unless it is of such extent as to cause severe dyspnea or other urgent symptoms. Even then, only 20 or 30 c.c. at the most should be withdrawn at a time. Sometimes the re-injection of 10 c.c. of the fluid into the subcutaneous tissues of the chest wall (autoserotherapy) every other day, repeating several times, will stimulate the absorption of the effusion.

In hemoptysis the patient should be placed in a semi-sitting position for the reason that expectoration, administration of medicines and the care of the patient during vomiting are facilitated. In bleeding of small amount, the above position and a few words of encouragement are all that is necessary. Hemorrhages of larger amounts require, in addition, the administration of 5 to 10 grams of table salt or 2 to 3 grams of sodium bromide three or four times a day. In extremely nervous patients or in cases with troublesome cough, morphine is an excellent remedy. If, in spite of these measures, the bleeding is very persistent or very copious it may be necessary to resort to artificial pneumothorax if it can be ascertained from which side the hemorrhage comes.

Just because a patient has tuberculosis is no reason why he cannot have other diseases; therefore one should be on the watch for non-tuberculous complications when the patient complains of unusual symptoms. Special attention should be given to the teeth, tonsils, eyes and nasopharynx and all regions that are likely to be the source of focal infection.

Education of the patient in all matters relating to tuberculosis is another essential point in the treatment of the disease. Tuberculosis cannot be cured without the coöperation of the patient and the patient cannot properly coöperate unless he has a good understanding of the disease.

To begin with, the patient should be informed that he has tuberculosis and that even though the prognosis may be favorable, he must not expect a cure within a few months. It is far better to tell him at once that, in all probability, the cure will be a matter of two or more years, than to give him the false impression that he will be well in two or three months only to be bitterly disappointed and perhaps very seriously discouraged.

Tuberculosis is one of the most curable of all diseases and one that most frequently relapses. The frequency of relapse, I think, is largely due to the neglect of properly educating the patient and to the spending of an insufficient length of time in finishing up the "cure."

BISMUTH IN SYPHILIS

The principles that apply to the selection of water-soluble as compared with oil-suspended insoluble preparations of bismuth in the treatment of syphilis would not seem to be essentially different from those now recognized for the use of mercury preparations except for the fact that bismuth preparations in general are pharmaceutically more eligible than are the corresponding mercurials. A water-soluble bismuth salt should be selected when a rapid transient effect is desired, and the administration of such a preparation should be more frequent than that of an oil suspended insoluble one. (Jour. A. M. A., June 22, 1929, p. 2123.)

THALLIUM POISONING

Three children died recently in London from poisoning by thallium acetate administered for ringworm of the scalp. This is an additional indication of the growing importance of thallium compounds as a dangerous poison. The first therapeutic use of thallium was to check sweating. Its action in causing a loosening and falling out of the hair was a "by-product" and most of our information about its other and more general poisonous effects in man has been obtained from its employment in epilation. Thallium is closely related chemically to mercury and lead. Although they appear earlier, the symptoms of chronic thallium poisoning are more like those produced by arsenic than by these other metals. (Jour. A. M. A., June 1, 1929, p. 1865.)

DIABETES MELLITUS AND ARTERIOSCLEROSIS*

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THE interrelationship between diseases of metabolism and diseases of the degenerative type is rapidly coming into the foreground in medical interest, particularly in connection with arteriosclerosis. To offer definite conclusions in this field is almost as difficult as to obtain undisputed observation in the field of constitutional types and diseases.

In clinical investigation, there are two main methods of approach: first, the statistical evidence about the frequency of the simultaneous occurrence of two or more definite pathological findings. This in biometrical terms is a demonstration of a high coefficient of correlation. It does not, however, tell anything in regard to the question of cause and effect. To throw light on the question of cause and effect, our second method is needed; *i.e.*, clinical information as to the time relationship between the occurrence of the different diseases. The relationship between diabetes and arteriosclerosis constitutes one of the most important problems of this type. Clinically, it is often difficult to distinguish the cause from the effect. This is particularly the case in the older diabetics where the onset of diabetes seems to closely coincide with the age of the development of arteriosclerosis.

In this communication the observation will be reported in a young man where the onset of diabetes mellitus preceded the symptoms of degenerative disease by ten years, and where our observations are unusually rich and dependable due to frequent hospitalization of the patient.

W. W., University Hospital, No. 30006 and No. 40924, white, male, age 33.

Family History.—Father living and well, 70 years old. Mother dead at 50, cause unknown. Six brothers and sisters living and well. One maternal uncle has diabetes, but is controlled on a qualitative diet.

Past History.—During his childhood he had measles and chickenpox and had two attacks of influenza which were very mild and he was frequently subject to colds. Otherwise his health was comparatively good until the age of nineteen, when he developed a series of boils and carbuncles. This continued with increasing severity until the age of twenty-two, when for the first time a urine analysis was made and the diagnosis of diabetes mellitus was made. His diabetes was controlled by diet without insulin. At the age of twenty-five he had a mild attack of influenza. At the age of twenty-six he developed a chancre, and for two years he was energetically treated for syphilis. At the age of twenty-eight his home was broken up and he was unable to live on his regular diet. He was soon forced to quit work because of loss of strength and symptoms of uncontrolled diabetes mellitus. This continued until the age of thirty, when he entered the University Hospital on August 9, 1924.

Physical Examination.—The patient is a well developed, well nourished man, thirty years of age, the picture of health. Height 5 feet 9 inches, pulse 90, respiration 18, temperature 98.6. The skin is warm and moist, with white spots about the body and neck, the result of numerous boils. Head normal, eyes normal, pupils react normally. Mouth—considerable gingivitis, pharynx reddened, tonsils small but inflamed, neck normal. Chest: normal shape, expansion good, no lagging, no deformities. Lungs within normal limits. Heart: apex beat not seen, but felt at the fifth interspace inside the midclavicular line. No palpable thrill present. Heart within normal position and size; no murmur heard. Radial arteries fairly easily collapsible. Blood pressure 135/88. Abdomen within normal limits. Extremities — muscular strength good, no spasms. Joints normal. Reflexes within normal limits. Diet on admission was: carbohydrate 40, protein 30 and fat 90. This was gradually increased until he was receiving carbohydrate 80, protein 50 and fat 150 with 10 units of insulin. Insulin was gradually discontinued and he was discharged on the same diet without the use of insulin.

Laboratory Examination.—Urine—specific gravity varied from 1.005 to 1.018, 1+ albumin, sediment contained a few white blood cells, otherwise negative. Acetone and diacetic acid absent. Blood sugar on admission .405 mgm.; lowest was .178 mgm. Blood urea nitrogen 14, creatinine 1.3, Wassermann negative.

After his discharge from the hospital he continued his usual occupation and was gradually able to go on a qualitative diet. In 1926 he noticed that his ankles were slightly swollen in the evening. This continued to increase in severity until the early part of July, 1927, when he was forced to stop his work because of the marked edema, and by the 13th of July it was necessary for him to be confined to bed. At this time he noticed he was getting rather dyspneic. He did not improve with rest in bed but became progressively worse. He was readmitted to the University Hospital July 30, 1927.

*From the University Hospital, Department of Medicine. These cases are published through the courtesy of Drs. Hilding Berglund and Henry L. Ulrich.

Physical Examination.—Middle aged man lying uncomfortably in bed. Temperature 98.6, pulse 90, respiration 20. Face somewhat puffy, numerous pigmented scars over the chest, neck and body. There are numerous brownish red ulcerations over his body which Dr. H. E. Michelson diagnosed as pyoderma. No ulcerations on the extremities. There is marked edema of the legs. Edema of the genitals and moderate edema of the back. Eyes: pupils regular, react to light and accommodations. Nostrils wide, scars of small ulcerations in the left nostril. Tonsils small, pharynx and neck normal. Lungs—moist râles in both bases; evidence of free fluid in the right base. Rhythm regular except interrupted by occasional extrasystoles. First and second sounds are distinct at the apex. Heart: Ml. 12.5 cm., Mr. 6.5 cm., the arch 3.8 cm. There was a systolic murmur heard at the apex. Blood pressure 130/110. The abdomen is sufficiently distended to suggest moderate ascites and there is shifting dullness. Liver and spleen not palpable. Rectal examination negative.

Ophthalmoscopic Examination.—Examination by Dr. F. E. Burch revealed complete obliteration of numerous fine arteries in the right eye and complete compression of a big vein in the left eye, causing thrombosis of this vein. There was a marked beading of the arteries and there was marked arterio-venous crossings. There was a spot of white, interpreted as diabetic exudate.

X-ray Examination. — *X*-ray of the extremities showed a moderate calcification of the arteries of the legs and arms. A six foot plate of the heart showed a marked generalized dilatation of the heart. Cardiac measurements were: transverse thoracic 29 cm., Ml. 12.5 cm., Mr. 6.5 cm., total 19 cm., longitudinal 12 cm., arch 3.8 cm.

Laboratory Findings.—Urine specific gravity varied from 1.014 to 1.028. On admission 8 per cent sugar (which gradually cleared up with treatment), many hyaline casts, many red blood cells, occasional leukocytes and positive acetone and negative diacetic. The acetone gradually disappeared on treatment. He was made sugar-free, but microscopic examination of the urine remained practically the same. His blood was 70 per cent hemoglobin, 4,620,000 red blood cells, 10,650 white blood cells, p.m.n.'s 84 per cent, lymphocytes 14 per cent and monocytes 2 per cent. Stool examination was negative. Blood sugar varied from .130 mgm. to .2 mgm. Blood urea nitrogen varied from 49 to 75 just before death. Fluid by thoracocentesis: specific gravity 1.015, albumin 1.5 per cent, 24,000 white cells per 1 cm., red blood cells 7,000. Guinea pig inoculation negative. Wassermann negative.

Electrocardiogram Examination.—Electrocardiogram presented the findings diagnosed as coronary disease, arborization block and extrasystoles.

Diet.—His diet while in the hospital was 50 carbohydrate, 50 protein and 120 fat. He remained sugar free without the use of insulin.

In spite of the use of various diuretics and digitalization he continued to run a course of cardiac decompensation and died August 28, 1927.

Clinical Diagnoses.—Arteriosclerosis, diabetes mellitus, myocardial failure, arteriosclerosis, coronary disease with thrombosis, pericarditis, generalized anasarca, latent syphilis, and benign hypertension.

Postmortem Examination.*—The body is that of a well developed, well nourished white adult male, 178 cm. in length and weighing about 160 lbs. Rigor is present; there is purplish hypostasis posteriorly; edema is noted in the face, arms, both legs, scrotum and penis; slight cyanosis is present in the lips; there is no jauundice. Each pupil measures 3 mm. in diameter; they are equal and regular. Numerous skin lesions are noted on the arms and chest. These are necrotic in the center, measure 1.5 to 3 cm. in diameter.

The peritoneum is smooth and glistening and the cavity contains about 2 liters of straw-colored fluid. The appendix is anterior and free. The diaphragm is at the fourth interspace on both sides. The right pleural cavity contains about a liter of cloudy fluid in which there are many flakes of fibrin. The left cavity contains about one liter of straw-colored fluid. The pericardium is smooth and glistening except on the right side, where it is covered with fibrin. The cavity contains about 200 c.c. of fluid in which are many flakes.

The heart weighs 440 grams. The myocardium is soft and pale. Both ventricles are dilated. The left ventricle shows a considerable amount of hypertrophy. The descending branch of the left coronary is calcified and the wall is markedly thickened. The lumen in places is almost obliterated. The right coronary artery shows extensive thickening but of less degree than in the left. About 7 cm. from the orifice of the right coronary the artery is thrombosed. A thrombus is present in the appendix of the right auricle. There is an extensive thrombosis in the apex of both ventricles. These thrombotic areas extend out into the lumen of the heart; they are smooth and white. No changes are noted in the valves. The root of the aorta shows many raised yellowish areas.

The right lung weighs 700 grams. The pleura is greatly roughened with a fibrinous exudate. Crepitation is much reduced in the middle and lower lobes. Both these lobes are solid in their posterior parts. On section pus is easily expressed from the region of the bronchioles. The smaller bronchi are somewhat dilated. The left lung weighs 480 grams. The posterior part of the lower lobe shows a condition similar to that found in the right lung.

The spleen weighs 205 grams. It is firm in consistency and purple in color. On section the pulp is found to be firm.

The liver weighs 1,860 grams. Its lower border extends 6 cm. below the costal margin in the right midclavicular line and 7 cm. below in the midline. The liver is firm in consistency, and on section the centers of the lobules show extensive darkening. No changes are noted in the gallbladder or gastro-intestinal tract.

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*Pathologist, Dr. B. J. Clawson, Prof. of Pathology, University of Minnesota.

The pancreas appears normal. The vessels do not show extensive arteriosclerosis. No changes are noted in the adrenals.

The right kidney weighs 105 grams; the left 115 grams. The capsules strip with some difficulty and leave surfaces containing many fine pits. On section the cortices show only a slight degree of narrowing. The renal arteries show marked sclerosis, so that the lumina are markedly reduced. Microscopic examination shows in many of the glomeruli a hyalinized condition of the arterioles with marked sclerosis. The mucosa of the bladder is smooth. There is no enlargement of the prostate.

The abdominal aorta shows quite extensive intimal thickening with several ulcerated areas.

The head was not examined.

Diagnosis—

1. Cardiac hypertrophy (hypertension)
2. Coronary sclerosis and thrombosis
3. Mural thrombosis
4. Pleuritis
5. Pericarditis
6. Bronchopneumonia
7. Hydrothorax, hydroperitoneum, edema of scrotum and generalized anasarca
8. Generalized arteriosclerosis
9. Arteriosclerosis of kidneys
10. Multiple skin lesions (pyoderma)
11. Diabetes, clinical

SUMMARY

This patient suffered from diabetes for fourteen years and at the age of thirty-three died from coronary disease with marked arteriosclerosis. His diabetes became less severe the longer he lived. His arteriosclerosis developed very markedly between the first and second periods of hospitalization. Dr. E. P. Joslin has shown that formerly 61 per cent of the fatalities in diabetics were due to coma, but since the introduction of insulin this has decreased to practically zero. Coincidentally arteriosclerosis as the cause of death has risen to 47 per cent. He expresses the thought that diabetics become prematurely old as evidenced by the increased arteriosclerosis. Bowen and Koenig have found in their series of cases arteriosclerosis demonstrable by the roentgenogram in 63 per cent in sixty-two observations in patients beyond the age of 40. At the same time they show that in 121 non-diabetic subjects in a similar age period, 28 per cent showed arteriosclerosis. From their work it appears that the cases of diabetes which have not been under control are more prone to the development of arteriosclerosis. Dr. Raymond Peterson in his study of the postmortem material at the University of Minnesota found that arteriosclerosis is much more pronounced in both severity and frequency in diabetics than in non-diabetics in the same age group. Sclerosis of the aorta was always present in diabetics over fifty years of age. Graded with 4 as the maximum, the largest number of cases falls in grades 3 and 4 in diabetics over 60 years of age; in non-diabetics the same age, the largest number fall in grade 1. Dr. Morris Nathanson studying the same material showed that 54 per cent of the cases with a history of diabetes had coronary disease, while approximately 13 per cent of the diabetics showed coronary disease.

It is interesting to note that the pancreas in this patient appeared normal and that the vessels did not show very extensive arteriosclerosis. Whether or not there was any regeneration during the fourteen years that he suffered from diabetes, it is hard to state, but at least no atrophy was present. His pancreas was within normal limits. What effect the anti-luetic treatment had in producing the marked arteriosclerosis is not known. Some have claimed that the heavy metals used in anti-luetic treatment predispose them to premature arteriosclerosis.

CONCLUSIONS

1. This case demonstrates how difficult it is to determine the relationship between diseases of metabolism and diseases of the degenerative type.
2. It does not seem necessary to assume that syphilis played a very important part in this case.
3. I present this as a case of juvenile arteriosclerosis, realizing that the relationship between diabetes and arteriosclerosis has not been determined. The diabetes, arteriosclerosis and syphilis might have contributed factors causing the untimely death of this patient.

BIBLIOGRAPHY

1. Bowen and Koenig: Bulletin of the Buffalo Gen. Hosp., Vol. V, No. 2, 1927.
2. Joslin, E. P.: Treatment of diabetes mellitus, 4th Ed., Sec. 10.
3. MacCallum, W. G.: Arteriosclerosis, Physiol. Rev. 2: (Jan.) 1922.
4. Nathanson, Morris. To be published.
5. Peterson, Raymond: Thesis for M. A., June, 1927. University of Minnesota, Dept. of Pathology.

CARDIAC DECOMPENSATION AND ITS TREATMENT*

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THERE is no disease today which has so large a morbidity and which results in so great a mortality as heart disease. It is the most important problem confronting the medical profession and touches practically every field of medicine. The prevalence of this disease and the mortality resulting from it are indicated by the fact that according to conservative estimates 2 per cent of the total population of the United States are seriously affected by it, and that it contributes 15 per cent of the total number of deaths from all causes. Charts I, II, and III reveal the significant fact that the heart death rate curve far exceeds all other rate curves, and that the rate is progressively rising.

The term heart disease is an inclusive term which covers any abnormal condition of the heart from the time the organ first reacts to an injurious agent or condition. In the earlier stages this is entirely without symptoms and may be also without signs. When, however, the pathological process has progressed to the point where the organ is unable to maintain an adequate circulation of the blood during the usual exertion and activities, thus producing certain sensations of distress or discomfort in the patient, we term such a condition heart failure. This inability to maintain an adequate circulation results in a disturbed blood distribution: A deficiency on the arterial side, and, what is even more important, a concomitant excess on the venous side. All the signs and symptoms which the patient presents—dyspnea, cyanosis, orthopnea, a painful and engorged liver, tachycardia, edema, ascites—are simply the result of an accumulation of blood under increased pressure on the venous side and at the same time a corresponding depletion in the arterial system. This concept of a disturbed blood distribution with an increased venous pressure as being the essential factor in heart failure cannot be emphasized too strongly. That is what congestive heart failure signifies. There is one exception to this definition, and that is the angina syndrome of the so-called angina pectoris where the patient dies suddenly from ventricular fibrillation without any signs of congestive failure. The anginal syndrome is thought to be principally the result of anoxemia of portions of the heart musculature caused by coronary disease (sclerosis, thrombosis) together with coronary spasm. Due to the blood deficiency and consequent lack of oxygen there is incomplete oxidation of metabolic products, resulting in an accumulation of lactic acid. When the acidity reaches a certain degree of hydrogen ion concentration, fibrillation sets in.

When the condition has further advanced to the point where the heart is unable to efficiently propel the blood through the vascular system except when the patient is restrained from doing any kind of work whatsoever, we designate such a condition as cardiac decompensation. This condition later progresses to the state when the above mentioned symptoms and findings are present even when the patient is confined to his bed at complete rest.

To summarize, these are the three stages of heart disease: First, when the affection is early and usually unrevealed; second, when the condition becomes apparent through various clinical manifestations brought on by strain or excessive exertion, such a condition being termed heart failure; and thirdly, when the signs and symptoms are present though the patient is restrained in his exertions or even at complete rest. This third stage is designated as cardiac decompensation.

PHYSIOLOGICAL PROPERTIES OF HEART MUSCLE

In order to properly understand heart disease and its sequences it might be well to recall the five intrinsic properties of heart muscle so well described by Gaskell and Engelmann. These are: stimulus production, rhythmicity; stimulus conduction; excitability; contractility; tonicity.

By *stimulus production* we mean that a stimulus material (hormone?), a chemical compound recently isolated by Haberlandt,[3] is continuously manufactured normally in the sinus node which regulates the rhythm activity of the heart. The

*Presented before the Minnesota State Medical Association, St. Paul, Minnesota, May 14, 1929.

CHART I

DEATH RATES PER 100,000 FROM SEVEN PRINCIPAL CAUSES OF DEATH
NEW YORK STATE EXCLUSIVE OF NEW YORK CITY, 1918-1926

Cause of Death	1926	1925	1924	1923	1922	1921	1920	1919	1918
Diseases of the Heart	303	273	261	270	260	234	243	221	247
Cerebral Hemorrhage	126	120	131	128	132	128	127	123	126
Cancer	121	121	120	117	115	114	109	106	104
Nephritis	121	118	112	111	111	110	117	116	128
Pneumonia	117	98	92	115	109	85	138	129	293
Accidents	86	89	88	90	82	76	78	76	88
Tuberculosis	85	89	91	95	95	99	112	126	145

sinus node is thus known as the pace-maker. Other regions of the heart have also the property of producing this stimulus material but to a much less degree. The formation of this material may be heightened or depressed by certain nervous influences and also by the amount and character of the blood supply.

Conductivity is the property of the heart muscle which makes it capable of transmitting stimuli from muscle cell to muscle cell. Conductivity can be heightened and depressed by nervous influences and by certain physical and chemical agents.

Excitability is that property of the cardiac muscle by virtue of which it responds to stimuli. It is measured not by the intensity of the action resulting from a stimulus but by the strength of the stimulus necessary to produce the contraction. Nervous and nutritional influences may increase and decrease excitability.

Contractility is the property by virtue of which the muscle cells become shortened, which is an important factor in emptying the heart chambers of their contents. Contractions of the heart muscles are always maximal. Contractility is measured by the degree of its contraction.

Tonus is that property of the heart muscle which enables the heart wall to resist stretching during diastole. This property maintains the efficiency of the heart by resisting over-filling of the chambers and by promoting a natural tension which is conducive to an effective contraction.

PATHOLOGIC PHYSIOLOGY

Disturbances of some of these properties of the cardiac muscle occur in practically all forms of heart failure, for in all affections of the heart, valvular, muscular or rhythmic, the condition of the muscles has to be taken into account. And it is important to bear in mind that in considering heart disease we are altogether too prone to stress the anatomic lesions, either gross or microscopic, and overlook the importance of certain physico-chemical changes in the muscles which though not demonstrable anatomically may result in muscular exhaustion and fatigue and may lead to a progressive impairment in function. Take as an example the myxedema heart, which is caused by some functional loss of muscle tone as a result of thyroid deficiency. This heart may enlarge, decompensate and proceed on a downward course until appropriate thyroid treatment has been instituted. Through such treatment there is usually a most remarkable recovery. Not only do the symptoms of decompensation disappear but the cardiac outline decreases and often recedes to normal proportions. Such a result is possible only in cases of functional disturbances and never where organic changes such as fibrosis and true hypertrophy are involved.

Disturbances of the last two properties of the heart muscle, contractility and tonicity, mentioned above, affect the functional capacity of the heart and may lead to cardiac enlargement and heart failure. Tonicity is the property which maintains the muscles in a state of static contraction, the impairment of which results in dilatation and also in hypertrophy. In this condition the heart does not empty itself completely during systole so that there remains a gradually increasing amount of residual blood. In the effort to compensate, the muscles finally are exhausted from overwork.

Disturbances of the other important property, contractility, are brought about chiefly by impoverishment of the circulation as in coronary disease. Broadbent[1] believes that angina pectoris is a result of a depressed contractility, and that pulsus alternans is a characteristic sign of

this, while precordial pain is a characteristic symptom. Precordial pain on exertion is a cardio-sensory reflex warning, he believes, signifying that the heart has reached the limit of its capacity and can no longer cope with the increased demand. These two factors just described, loss of tonus and depressed contractility, are the most important ones involved in myocardial heart disease. They are also more or less important in the other forms of heart failure.

CHART II

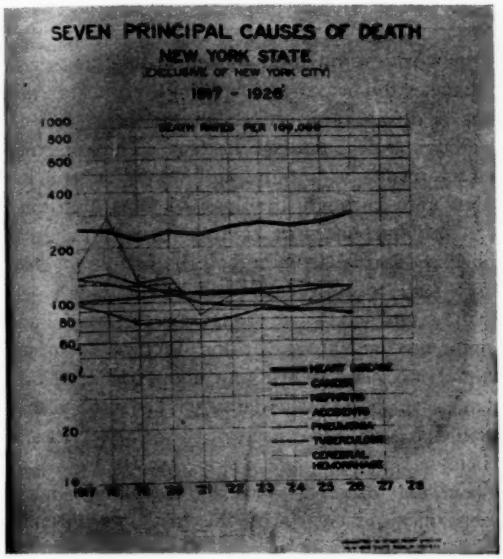
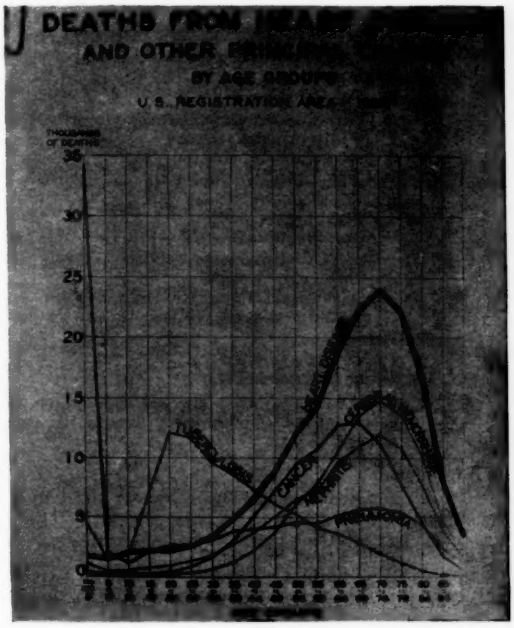


CHART III



TYPES OF HEART DISEASE

According to Chart IV it will be seen that the efficiency of the heart may be impaired by three main types of lesions acting singly but often in combination, which may ultimately lead to cardiac failure: (1) Damage to the muscle; (2) disturbances of the valves; and (3) disorders of the rhythm.

Myocardial disease.—This type contributes by far the greatest number of cases of heart failure, variously estimated from 65 to 77 per cent. This class is composed principally of hypertensive cardiovascular disease and includes practically all types of heart disease free from valvular involvement. Chart IV, Group 1, shows in detail the different types of heart disease that go to make up this group. As already stated, essential hypertension and coronary disease furnish by far the greatest number of cases, the remainder being furnished by syphilis, goiter, toxic states and myocarditis. Myocardial heart disease is most often incorrectly diagnosed as chronic myocarditis, a condition which pathologically is extremely rare. The classification suggested in Chart IV is of value since it attempts to unite the underlying pathological conditions with the various types encountered clinically. From 80 to 95 per cent of this group occur in individuals over 50 years of age. Chart X shows the relative frequency of the different types of heart disease.

There is still some doubt in the minds of many as to the occurrence of heart failure through hyperthyroidism in otherwise normal hearts. Studies of the literature reveal the fact that careful pathological investigations have been made on patients dying from cardiac decompensation associated with hyperthryoidism and that many of these cases show gross and microscopic evidence of acute myocardial damage. Thacker[4] reviews the literature on the subject and concludes that from 2 to 6 per cent of cases of hyperthyroidism present severe cardiac damage brought about in approximately the following manner: increased metabolism increases the car-

CHART IV

Classification of Heart Disease

I. *Myocardial*
- A. Acute exudative myocarditis
  1. Abscesses
  2. Infected mural thrombi
  3. Extension from pericarditis
- B. Toxic myocardium
  1. Mild, in mild infectious processes
  2. Severe, in severe infectious processes, as diphtheria
- C. Chronic myocarditis—syphilitic myocarditis (Warthin) rare
- D. Chronic adhesive pericarditis, usually with adhesive mediastinitis
- E. Hypertension heart
  1. Without coronary disease
  2. With coronary disease
  3. Secondary to chronic glomerulo-nephritis
- F. Coronary disease; with or without hypertension
  1. With hypertrophy
     a. Without congestive failure
     b. With congestive failure
  2. Without hypertrophy
     a. Without congestive failure
     b. With congestive failure
- G. Thyroid heart
  1. Hyperthyroidism
  2. Myxedema heart
- H. Right heart failure; chronic emphysema, adhesive pleuritis, pulmonary fibrosis, Ayerza's disease.

II. *Valvular*
- A. Endocarditis—principally mitral and aortic
  1. Rheumatic
     a. Acute
     b. Recurrent
     c. Chronic old valve defect
     d. Pancarditis, mostly rheumatic, with endocarditis and pericarditis
  2. Bacterial
     a. Acute—primary, secondary
     b. Subacute (Libman)
        (1) Primary on normal leaflets
        (2) Superimposed on old valve defects
- B. Chronic valvular disease, non-rheumatic
  1. Congenital heart disease, principally pulmonary lesions with or without other heart anomalies
  2. Atherosclerosis, degenerative, senile
- C. Syphilitic, aortic

III. *Disorders of Rhythm*—Infrequent as primary cause of heart failure
- A. Auricular fibrillation (and flutter)
  1. Primary, paroxsymal
  2. Secondary
     a. Mitral stenosis
     b. Hypertension heart
     c. Hyperthyroid heart
     d. Other conditions
- B. Paroxysmal Tachycardia
  1. Auricular
  2. Nodal
  3. Ventricular
- C. Heart Block
  1. Partial
  2. Complete—with or without Stokes-Adams syndrome, usually secondary to coronary disease and myocardial fibrosis

CHART V

Principal Causes of Cardiac Deaths

1. Hypertension heart
2. Valvular disease
3. Coronary disease—anginal syndrome
4. Syphilis of heart and aorta
5. Pericarditis, pancarditis
6. Congenital heart disease
7. Myocarditis, acute and chronic
8. Right heart failure, from intrapulmonary lesions
9. Thyroid heart—hyperthyroidism, hypothyroidism
10. Disorders of rhythm

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diac work, stimulation of the sympathetics also increases the heart rate, and the thyroid secretion has a direct toxic effect on the heart muscle, leading to degenerative lesions. The combined effect results in fatigue and dilatation and hypertrophy through the loss of muscular tone. Later disturbances of rhythm, usually auricular fibrillation, may set in and all the factors together may lead to progressive dilatation and hypertrophy and finally to congestive heart failure. Both mechanical and toxic factors therefore come into play in bringing about the final outcome. A previously damaged heart of course would hasten the onset of circulatory insufficiency.

Valvular disease.—Valvular lesions impair the mechanical efficiency of the heart as a pump, thus increasing its work. However, the true functional capacity of the heart, being dependent on the integrity of the muscles, may remain unimpaired. The greatest etiological factor in valvular disease is rheumatic fever, which occurs most frequently between the ages of 15 and 25. After the age of thirty acute rheumatic fever is relatively infrequent. This lesion most frequently involves the mitral leaflets, which are affected to a greater or lesser degree in about 75 per cent of cases of rheumatic endocarditis. The most common lesion is mitral stenosis and regurgitation. The occurrence of a pure mitral insufficiency is extremely rare. The aortic leaflets are next in frequency of involvement, while lesions of the tricuspids and pulmonary valves are very rare. In older people sclerosis of the aorta and at times of the mitral valves are encountered, which may be either calcification of previously damaged valves resulting from rheumatic fever or less frequently may be a simple senile atherosclerotic process.

In the group of valvular diseases is included also aortic regurgitation associated with meso-

aortitis luetica. This lesion is eight times more frequent in males than in females. The characteristic feature of this lesion is that it produces a pure aortic insufficiency which is a result of dilatation of the aortic ring weakened by the syphilitic degeneration of the media. Very rarely is there any stenosis present. When this occurs it is the result of synechiæ of the cusps.

Valvular heart disease is much less frequent than is generally supposed. According to Cabot it comprises only 23 per cent of all causes of heart disease. To the laity and to many physicians as well, the term heart disease connotes valvular heart disease, which of course is a gross error. The mitral systolic murmur at the apex which is so often a relative murmur and the so-called functional pulmonic systolic murmur are largely responsible for the excessive number of clinical diagnoses of mitral regurgitation. The importance of properly evaluating cardiac murmurs is far from being academic. Too many patients with utterly unimportant cardiac murmurs in otherwise normal hearts are made temporary or often confirmed semi-invalids through some casual remark from a doctor that he has found a leaky valve. No amount of reassurance by one or many doctors can eradicate the feeling of invalidism so inculcated in an impressionable neurotic individual. This is especially true if the patient has some member of his family suffering from true heart disease. One must be very careful lest a patient leave the office in a worse condition mentally than when he enters.

Disorders of rhythm.—As a primary cause of heart disease this group is relatively unimportant. The disorders of rhythm are brought about by disturbances, functional or organic, in the first three of the five above named properties of cardiac muscle, stimulus production, stimulus conduction and muscular excitability. Auricular fibrillation is the commonest form of arrhythmia and is most often secondary to chronic mitral stenosis, hypertension and hyperthyroidism. Paroxysmal tachycardia, auricular flutter and heart block occur much less frequently. Heart block results from disturbances in conductivity, and the most severe form may lead to Stokes-Adams syndrome. Extra systoles, both ventricular and the less common auricular, do not come under this discussion, since these never lead to any serious symptoms.

CARDIAC ENLARGEMENT

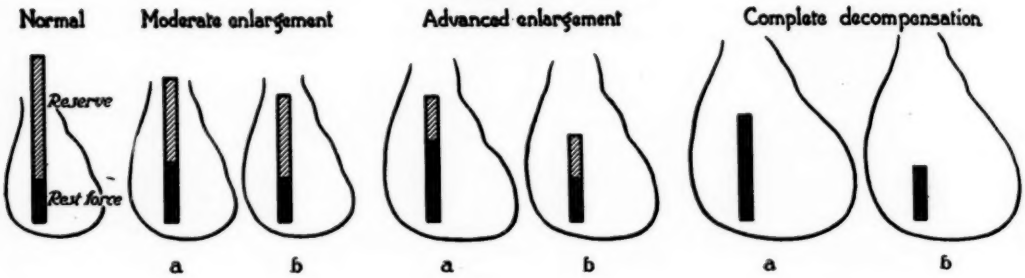
The most important criterion for establishing the presence of heart disease is cardiac enlargement. As will be seen later, there may be occasional cases of severe heart disease, even to the point of congestive failure, with no increase in cardiac outline, but such cases are rare. The cardiac outline as obtained through careful physical examination and checked by a roentgenogram is therefore extremely important in cardiac diagnosis. The shape as well as the size of the heart helps in determining within certain limits how far a given heart has progressed toward decompensation. For every heart, as soon as it shows definite signs of enlargement, has started down the path towards heart failure.

To thoroughly understand this fact, one must remember that the normal heart does not perceptibly increase in size no matter how great the demand placed upon it. This fact has been well established in various countries through careful studies on marathon runners and other athletes. In a recent study[2] of twenty-three long distance runners at the end of a race from Los Angeles to New York it was found that the hearts were actually smaller in thirteen, normal in five, and showed slight enlargement in five, in only one of which there was an increase above the average in the cardiothoracic ratio. The heart of a trained athlete may show some increase in weight through pure hypertrophy but this increases the cardiac shadow only by a few millimeters. It is only when the heart dilates that the cardiac outline shows definite enlargement. And a dilated heart is always a pathological heart. The normal heart responds to increased demands by an increase in the minute volume output brought about by an increased venous return of the blood which raises the venous pressure, filling the ventricle more fully and causing better and more complete systolic contraction. The systolic output is thus increased, which together with the increased pulse rate may raise the heart output from 4.5 liters per minute to as high as 14 liters. The upper range in this increased work is known as the limit of cardiac reserve.

When the venous pressure on the ventricle exceeds the physiological limit of the muscle, the muscle stretches and the contraction becomes weaker and less efficient. This happens when the heart has been altered through disease pro-

cesses. The over-stretching of the muscle gradually leads to permanent enlargement, chiefly dilatation. In a general way, lesions of the valves produce cardiac enlargements which differ somewhat from those secondary to hypertensive states in that there is less hypertrophy present.

CHART VI



a. Hypertension b. Other diseases

This diagram shows schematically the gradual cardiac enlargement resulting from heart disease. The total heart force is represented by the combined rest force and reserve force. In the hypertension heart (a), the total heart force is reduced proportionately less than in the other types but the cardiac reserve is no greater. This is due to the increased work of the heart with the patient at rest using up the cardiac reserve caused by the increased blood pressure. The diagram attempts to emphasize the fact that with the increase in the heart silhouette there is a corresponding decrease in the cardiac reserve. The last two views show very large hearts where the cardiac reserve has been entirely used up. Patients with hearts in this stage are bedridden in a state of complete decompensation.

It is thus seen that the presence of cardiac dilatation and hypertrophy signify that the reserve forces of the heart are being brought into play and it might be stated in general terms that the increase in cardiac outline develops at the expense of cardiac reserve. An enlarged heart is on the road to heart failure and the larger the heart the nearer it is to decompensation. Chart VI illustrates this idea. The normal heart may be considered to possess a heart force consisting of a small rest force and a large reserve force. As soon as the heart is unable to maintain an adequate circulation of blood without enlargement it starts drawing on its reserve force. This reserve force may be depleted in two ways, by a diminution of the total cardiac force or by an increase in the rest force. Often both factors are present. In essential hypertension the reserve force is spent even while the patient is at rest, because of the great increase in work due to the increased blood pressure. The total cardiac force is reduced relatively less than in valvular disease since there is a greater element of hypertrophy present. As the process goes on and the heart becomes very large the reserve force may be all used up and complete decompensation ensues. The size the heart must reach before the cardiac reserve is expended and the condition of decompensation sets in, naturally varies with different individuals and unfortunately no measurements or set formulæ can be stated to indicate the degree of loss of cardiac reserve present at any given time.

When the cardiac outline is found to be within normal limits one should be slow in making a diagnosis of serious heart disease, even though murmurs and suggestive symptoms may be present. The only exceptions to this rule are cases with anginal syndrome and cases with acute infections of the heart or advanced states of malnutrition when cardiac weakness and even decompensation may be present in hearts that show little or no increase in size. These cases, however, are not common. Chart VII shows some of the common errors in cardiac diagnosis as revealed by careful postmortem studies. In mortality statistics nephritis ranks high, occupying from second to fifth place among the principal causes of death. I believe that the frequency of nephritis is greatly exaggerated because many cases of cardiac decompensation associated with edema and albuminuria secondary to essential hypertension are being diagnosed as nephritis. A correction of this error would greatly lower the nephritis death rate and proportionately increase the death rate from heart disease.

CHART VII

Common Clinical Diagnoses (according to Dr. E. T. Bell)	Pathological Findings
1. *Chronic Myocarditis*	Hypertension Coronary sclerosis Old valve defect Adherent pericardium Toxic myocardium
2. *Mitral Regurgitation*	Hypertension Bacterial endocarditis Old valve defect Adherent pericardium Toxic myocardium Coronary sclerosis (congestive type)
3. *Aortic Regurgitation*	Bacterial endocarditis Old valve defect Luetic aortitis Hypertension
4. *Chronic Interstitial Nephritis*	Hypertension heart with congestive failure, edema and albuminuria Malignant hypertension

CRITERIA FOR DIFFERENTIAL DIAGNOSIS

There are certain criteria which may help greatly in the differential diagnosis of the various types of heart disease and these may be tabulated as follows.

I. Acute valvular disease, endocarditis
   1. fever (chills)
   2. leukocytosis
   3. toxemia
   4. rheumatic pains, signs of acute arthritis
   5. development of murmurs

In subacute bacterial endocarditis the following may be present in addition to the above:

   6. petechiæ and other embolic phenomena
   7. positive blood cultures
   8. enlarged spleen
   9. red blood cells in the urine
   10. anemia

Death may be due to toxemia, embolic accident or congestive heart failure.

II. Chronic endocarditis, chronic valvular defect
   1. history of previous attacks of rheumatic fever, tonsillitis, chorea
   2. enlarged and altered cardiac outline
   3. murmurs, the character depending on the type of valvular lesions, diastolic murmurs being the more important
   4. for mitral stenosis without fibrillation the presystolic murmur is important
   5. for aortic stenosis a palpable systolic thrill is important
   6. alterations in character of pulse
   7. auricular fibrillation especially in mitral stenosis
   8. anginal attacks in aortic lesions
   9. cardiac asthma principally in aortic lesions

Death occurs principally from congestive failure, acute endocarditis being occasionally superimposed. Mitral lesions may result in repeated attacks of decompensation followed by compensation, ability to compensate being good. Aortic lesions, when once decompensation occurs, show a very poor tendency to compensation.

III. Toxic myocardium
   1. follows acute infections, diphtheria, influenza
   2. electrocardiograph may show charteristic changes
   3. tachycardia on slight exertion
   4. complete restitution in practically all cases

IV. Thyroid heart
   A. Hyperthyroid heart
      1. symptoms of hyperthyroidism
      2. increased metabolic rate
      3. tachycardia

4. auricular fibrillation
5. symptoms of congestive failure
6. pulsus alternans, serious
7. treatment of thyroid condition often results in cure

B. Myxedema heart
1. symptoms of myxedema
2. low basal metabolic rate
3. generalized enlargement of the heart
4. symptoms of congestive heart failure
5. thyroid extract restores condition to normal, functionally and anatomically

V. Hypertension heart
1. generally in individuals past 40 years of age
2. blood pressure high, may be low
3. moderate to marked cardiac enlargement, aortic in type
4. retinal changes
5. often familial history
6. altered heart tones especially when associated with coronary disease
7. precordial distress, anginal attacks
8. cardiac asthma, nocturnal dyspnea
9. auricular fibrillation
10. cerebral accidents, apoplexy in about 20 per cent
11. mitral systolic murmurs, relative in origin
12. aortic diastolic murmur, very rare
13. if decompensated, poor comeback, only slightly better than aortic valvular disease
14. uremic death from arteriolar sclerosis of the kidneys in about 8 per cent of cases

VI. Coronary disease
1. age usually over 50, may be as early as 30
2. anginal syndrome common; crowding, retrosternal and precordial pressure pain with or without radiation
3. electrocardiogram frequently characteristic
4. mild diabetes, often
5. altered muffled heart tones
6. may be with or without hypertension
7. with or without enlargement
8. with or without congestive failure
9. long-continued pain generally signifies coronary thrombosis, serious
10. paroxysmal dyspnea and cardiac asthma
11. arrhythmias
12. death may be sudden from ventricular fibrillation

VII. Syphilitic cardiovascular disease
1. history of lues
2. usually above age of 40
3. Wassermann positive in about 70 per per cent
4. pure aortic regurgitation
5. dilatation of aorta, evidence of mesoaortitis luetica
6. aneurysm, frequently
7. anginal syndrome, principally from closure of ostia of coronaries
8. congestive heart failure

VIII. Congenital heart disease
1. cyanosis from infancy
2. clubbed fingers
3. atypical murmurs at base
4. polycythemia
5. other congenital deformities

IX. Disorders of rhythm, primary
1. irregularities in pulse
2. abnormal electrocardiograms
3. prognosis generally good
4. termination of a few in congestive heart failure

CLINICAL EVIDENCES OF HEART FAILURE

The clinical evidences of heart failure are usually definite enough so that a correct diagnosis can be made by most physicians who are careful in the examination of the patient. An important fact to bear in mind is that the stethoscope gives practically no information about compensation or decompensation and one should therefore not diagnose with the stethoscope. A careful history is most essential in obtaining information relative to the ability of the heart to do work under different degrees of strain, since one is entirely dependent on the history for the subjective sensations so important in cardiac diagnosis. In the early stages some of the symptoms such as palpitation, undue shortness of breath, precordial pain, substernal crowding, fatigue, may follow

only violent exertion or exhaustion. Later, as the reserve force gradually diminishes, these same symptoms may be present after less strenuous effort. Pain, breathlessness and undue fatigue, together with symptoms referable to the stomach, are often early indications of a failing heart. As the condition progresses, dyspnea increases, there develops cyanosis, râles in the bases of the lungs, the liver becomes large and tender, and there may develop edema of the legs, ascites, hydrothorax, oliguria, paroxysmal dyspnea, tachycardia, cardiac asthma. The enlarged distended cervical veins show the increase in the venous pressure. In severe cases there may develop frothy blood-stained sputum signifying edema of the lungs, especially in mitral disease. A sudden onset of coughing with expectoration of bloody sputum often suggests pulmonary infarction, the embolus originating in a thrombus in the right auricle. Other sudden embolic phenomena such as hemiplegia, splenic infarct, kidney infarct (hematuria) are manifestations of emboli from left auricular thrombosis. Thromboses in the auricles signify either cardiac decompensation or auricular fibrillation. Thrombosis in the ventricles is quite rare and when it occurs is associated with advanced myocardial degeneration and fibrosis from coronary disease.

A low systolic blood pressure together with a low pulse pressure is a definite sign of a failing heart. Vital capacity is of value as a quantitative measure of the degree of heart failure. It rarely gives any evidence of cardiac insufficiency which cannot be estimated by other methods of investigation. The electrocardiograph often shows evidence of myocardial damage that cannot be estimated in any other way. This is especially true in cases of severe coronary disease, and in coronary thrombosis. The roentgenogram is a very valuable aid in cardiac diagnosis because it gives an accurate silhouette of the heart both as to shape and size and thus furnishes very reliable evidence of cardiac disease. The evidence of decompensation lies principally outside of the heart itself, as a result of increased venous pressure which is manifested in the signs and symptoms of passive congestion. A markedly enlarged heart signifies that the heart has used much, if not most, of its reserve force and that the outlook is not too favorable. If not much enlarged the heart may still have considerable reserve force. Gallop rhythm and pulsus alternans are very grave signs of heart failure and are evidences of serious myocardial damage.

CHART VIII

DEATH RATES FOR HEART DISEASE AND OTHER PRINCIPAL CAUSES OF DEATH, MINNESOTA DEPARTMENT OF HEALTH, GERDA C. PIERSON, DIRECTOR, 1928

Causes	Death Rates per 100,000
Heart Disease	150.70
Cancer	106.40
Pneumonia	66.09
Nephritis	53.78
Tuberculosis (all forms)	53.60
Influenza	39.79
Diabetes	18.81

(To Be Concluded in September)

Editor's Note.—Owing to the extensiveness of this paper it has been divided into two sections. Part II, taking up the matter of treatment, will appear in our next issue.

CASE REPORTS

SYPHILIS OF THE STOMACH*

REPORT OF CASE

J. A. JOHNSON, M.D.
Minneapolis

The patient, Mr. L. J., fifty-four years of age, came to the clinic on January 23, 1922, and gave the following history: He had been well until four years previous, when he began to complain of indefinite stomach trouble such as indigestion, belching of gas, and eructation of sour material. Three years previous he was taken with severe cramps after a heavy meal and for the first time vomited. Following this attack he gradually grew worse and for the past few months he had been able to hold practically nothing on his stomach. His weight two years previous was 145 pounds and at the time of examination was 112 pounds.

X-ray of his stomach showed almost complete obstruction at the lower third. There was a large palpable mass over the stomach area. The urine was normal. There was a slight trace of blood in the stool. No free HCl was found in stomach contents. The hemoglobin was 70 per cent, R.B.C. 3,900,000, Wassermann 4 plus.

Neurological examination was negative except that there was slight irregularity of his pupils. He had had his initial lesion nineteen years before. He had been well treated over a period of several years, both by internal and external medication.

We were unable to make a definite differential diagnosis in this case and because of his extreme emaciation felt that an exploratory was justified. He was operated in February, 1922, with the following findings: The stomach wall was about three times the normal thickness. There were gray striations, especially over the fundus of the stomach, which much resembled a linitis plastica or the so-called leather-bottle stomach. The hypertrophy was uniform, being, however, a little more marked at the pylorus. The stomach was carefully palpated anteriorly and posteriorly and there was no evidence of any localized area of infiltration. There were adhesions extending from the duodenum to the liver and this evidently had considerable to do with his obstruction. There was only one enlarged gland present at the pyloric end. The rest of the abdomen was normal. Posterior gastro-enterostomy was done and at the same time a large portion of all the layers of the stomach was removed for microscopic examination. A pathological report from Dr. Bell at the University of Minnesota stated that he was unable to find any evidence of malignancy in the stomach or gland. There was very extensive atrophy of the mucosa. The submucosa and muscular coats were very much hypertrophied. He stated further that this was quite a typical picture of what occurs in syphilis of the stomach. The postoperative convalescence was entirely normal. I saw him on November 28, 1925, at my office, three years after the operation. He said that he had had absolutely no trouble since. He received antiluetic treatment from his family physician for a couple of years. I saw his family physician in December, 1928, and he told me the man had been in splendid health ever since.

*Presented before the Minneapolis Surgical Society, March 7, 1929.

SYPHILIS OF THE STOMACH*

REPORT OF CASE

JAMES M. HAYES, M.D.
Minneapolis

Formerly, syphilis of the stomach was considered rare. Recent improved diagnostic methods, *x*-rays, serology, etc., have brought out the fact that it is not so rare. It has recently been estimated that it occurs once in about three hundred gastric lesions.

In 1905 Chiari gave the first classical description of this condition from post-mortem examination. Myers in 1912 reported fifty-eight cases from the literature. Smithies reported twenty-six clinical cases in 1915. In 1917 Eusterman reported twenty-three clinical cases. Downes, in 1917, reported eight clinical cases.

From these and other exhaustive studies the following definite facts have been brought out:

1. The condition may occur in congenital or acquired syphilis.
2. Gastric symptoms appear from four to twenty-five years after the initial lesion.
3. Duration of symptoms averages about eight years.
4. These are usually dyspeptic symptoms simulating cholecystitis, duodenal or gastric ulcer.
5. Pain is usually severe immediately after meals—frequently a cutting pain, worse at night.
6. Usually anacidity or achylia, probably due to destruction of mucosa, is present.
7. Pain is frequently relieved by alkalies, but not by food.
8. Marked weight loss may occur, without corresponding cachexia.
9. Marked deformity of the stomach occurs when symptoms become pronounced.
10. Usually a marked sclerosis develops in the mid-portion of the stomach, giving an hour-glass or dumb-bell deformity.

*Presented before the Minneapolis Surgical Society, March 7, 1929.

11. A palpable mass or six hour residue is frequently not present.

12. There is not a steady and continuous progress to fatal termination as in cancer of stomach.

13. Under specific treatment, symptoms usually clear up but the deformity remains.

CASE REPORT

A married woman aged 31 could give no history of a primary lesion. Her family history and previous personal history were negative.

In 1920 she first had severe attacks of epigastric distress which came on in the night. Some form of medication by mouth gave her relief, and she had no more marked symptoms until 1923. Then she had a sudden severe attack of epigastric pain with symptoms of perforation of an abdominal viscus. She was taken to the General Hospital, the abdomen opened and a gastric perforation closed. Following the operation she had epigastric distress and digestive symptoms, but no severe pain.

The patient was married in 1924 and gave birth to a healthy, normal child.

In 1926 she had a sudden, very severe epigastric pain which came on after eating. She was relieved for a short time by a hypo of morphine, but attacks recurred frequently. She was taken to the hospital and *x*-ray of the stomach showed a typical and extensive dumbbell deformity. The patient did not appear older than her age, and there was no marked cachexia. There was no six hour residue nor palpable mass. The blood showed a four plus Wassermann reaction. The patient was put on specific treatment and the symptoms disappeared. She had very little difficulty during the following two years.

About one year ago, April 4, 1928, the patient had a sudden, severe, gastric hemorrhage. She was brought to the hospital and transfused. The hemorrhage recurred. Transfusion was repeated four times but the hemorrhage continued and surgery was resorted to. Almost the entire anterior gastric wall had become adherent to the abdominal wall and there was a large ragged ulcer perforating posteriorly into the pancreas. The gastric wall for some distance from the ulcer resembled, in consistency, the so-called "leather bottle" stomach. The ulcer was excised together with a large part of the lower gastric wall, a high gastro-enterostomy was done and the wound closed without drainage.

The patient made a smooth recovery and has had practically no trouble since. *X*-ray of the stomach now appears almost normal.

PRESIDENT'S LETTER

AUGUST, the real vacation month, is just in the foreground. The busy, hardworking doctor owes it to himself, his family and his patients to take a rest and occupy his mind, as well as his body, with interests that are quite foreign to his daily duties. To get away and find solace and surcease in the great outdoors is the panacea for some of us. Others will visit distant cities to observe the activities of men engaged in pursuits similar to our own and to refresh and replenish our store of knowledge as to the progress and development of things ever new. To each in his own way the results of a vacation are shown in an added zest for better things to follow, a deeper insight into human nature, a desire to help and give comfort, to instil courage into weary and discouraged minds and bodies, and to start again that wish to achieve and accomplish, temporarily lost or abandoned.

For those who long to get away from the haunts of men and bask in the sunshine of the wilderness Minnesota offers opportunity unlimited.

August is to be noted as a month fraught with much of interest and value to the profession of medicine. In August, 1897, Sir Donald Ross first announced his far reaching discovery of the fact that the dapple-winged mosquito was the carrier of malaria. In August, 1904, General Gorgas began his epoch-making assault on the mosquitoes of the canal zone, making this area a safe place for the white man to live, basing his activities upon Sir Donald's researches, and this other species of mosquito responsible for the spread of yellow fever was virtually exterminated and instead of a death rate of seventy-five per cent of the workers who labored in the canal zone for nine years under the French, the death rate was reduced to almost zero from this scourge. In August, 1914, just seventeen years later, the canal was completed and opened for shipping. It is interesting to note that in August, 1774, Priestly, a famous chemist of his time, discovered oxygen. He called it pure air from the fact that when inhaled it produced a feeling of exhilaration and vastly increased the vigor and volume of a candle flame.

Whatever we do or wherever we spend our vacation days let us ever keep in mind that we are attempting to store up surplus energy to carry us through the trying days ahead.

President,
Minnesota State Medical Association.

EDITORIAL

MINNESOTA MEDICINE

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THE *X*-RAY FILM HAZARD

Although some two months have passed since the tragedy in the Cleveland Clinic, very little locally has been accomplished in a constructive way to prevent the recurrence of a similar tragedy. While for the most part the storage of *x*-ray films in comparatively large quantities is being carried out in this locality in conformity with the regulations specified by the Fire Underwriters, there are exceptions. It is said that at the Crile Clinic these specifications had been complied with, which indicates the need for their revision.

A perusal of the report on the Cleveland Clinic fire by the National Board of Fire Underwriters and the Ohio Inspection Bureau shows that the real cause of the fire will never be definitely known. There was a leak in a steam pipe in close proximity to several tons of stored *x*-ray films which conceivedly might have heated some films to the point of decomposition. According to the report the more generally accepted cause of the fire was the heat from an incandescent lamp in the storage room, and the possibility of a cigarette having started the fire is to be considered. The basement position of the room with the easy access of gases to the air shafts leading to all parts of the building was a particularly unfortunate setting which made the outcome so tragic.

The early rumors that the deadly fumes were from a source other than the films were disproven. The exact nature of the gases is not known. The elements carbon, hydrogen, nitrogen, and oxygen were contained in the films and the report estimates that the poisonous gases probably consisted of about 30 per cent carbon monoxid and about 30 per cent nitrogen peroxid, which is also very poisonous, exerting a corrosive action on human tissues. Hydrogen was probably present and the explosions doubtless resulted from the various gases being heated above their ignition point so that they flashed on contact with air.

The fire hazard of *x*-ray films has long been known. Certain precautions have been specified by fire underwriters, and various municipalities —Minneapolis, for instance—have stringent rules for storage of *x*-ray films. Some municipalities have had no special regulations to cover *x*-ray film storage and to date have adopted no satisfactory regulations. It is true that as a fire hazard *x*-ray films stored in small quantities may not be so great. There was evidence, however, in the Cleveland Clinic building that the gases evolved produced a hot flame. With their explosive possibilities and the danger of evolving lethal gases, their menace to human life is great. Possibly life insurance companies

will have to be depended upon to procure proper action.

The report mentioned proposes two reasonable methods of assuring proper safeguards in the storage of films. One is the compulsory substitution of a type of film which does not possess the dangerous qualities of the nitrocellulose film commonly used. The other is the adoption by states and municipalities of stringent requirements covering storage and handling of the present films. Storage in outside buildings and well removed from other structures such as the method used at the Mayo Clinic is preferable. Where this is not feasible the regulations of the National Board of Fire Underwriters, which require outside vents, automatic sprinkler protection, and so forth, should be carried out.

The Public Health Council of New York state, acting on the recommendation of a special committee appointed by Acting Governor Lehman, has prohibited the sale and distribution of the ordinary nitrocellulose films in the state except in New York city, effective Sept. 1, 1929. Similar action will doubtless be taken in New York city also. This means that in New York the acetate films will replace those now in use.

It has been said that the detail furnished by the acetate films is inferior. They are more expensive owing to certain difficulties in manufacture. Certain large hospitals in the East, however, have used them exclusively for some time. It is rumored that the acetate films are likely to be made as efficient as the dangerous ones. That being the case the slight additional cost should not prevent their substitution.

THE TEACHING OF MASSAGE

Massage, mechanotherapy and physiotherapy being adjuncts of the treatment of the sick, the medical profession is interested in these subjects. Manual massage formerly was a profession in itself. Masseurs in this country at least have added mechanotherapy and in many instances physiotherapy to their armamentarium. Just how far the medical profession should go in backing the formation of an independent school by masseurs to teach these subjects is a question. It is felt by the Council on Physiotherapy of the American Medical Association that the teaching of physiotherapy should be carried on in connection with an established medical institution as is done at Harvard and Columbia universities.

While the State Board of Medical Examiners now has charge of the licensing of masseurs in Minnesota, this Board must, according to the law and quite naturally, depend on professional masseurs in determining eligibility of applicants. Whether a group of physicians should act as an advisory board to have supervision of the conduct of a school of massage, mechanotherapy and physiotherapy combined is open to some question. We should be inclined to recommend that the teaching of these subjects be made a part of established educational institutions.

The outline of a proposed independent school of massage, mechanotherapy and physiotherapy to be established in the Twin Cities was given publicity in the "President's Letter" in the July number of MINNESOTA MEDICINE. This should not be construed as an official endorsement of the project for no official action has been taken nor is likely to be taken by the State Medical Association. The name "Basic Science Institute and Sanatorium" proposed for the new school is misleading and consequently ill chosen.

The practice of massage as a profession in this country has not had the attention nor development that it has had elsewhere, for instance in the Scandinavian countries. Possibly there is need for a good school in this country and in our own state.

OZONE AND VENTILATION

The ill effects from lack of ventilation do not arise from any contaminating substance in the air but are due to the physical properties of the air—chiefly its lack of cooling power. "Bad air" is made so by being too warm, too moist or too stagnant. While ozone has been suggested as a means of neutralizing the carbon monoxide in the air, particularly in garages, this is based on the misconception that it combines with carbon monoxide in low concentrations. Its use in garages is inadvisable, as ozone, by its odor, will give a feeling of false security and thus foster a tendency to cut down ventilation. There has been no sound scientific work brought forward to show that there is any place whatever for ozone in the problems of ventilation; on the contrary the inefficacy of ozone generators has been demonstrated by careful scientific experiments. (Jour. A. M. A., June 29, 1929, p. 2186.)

OBITUARY

Charles Stanley McVicar

1880–1929

Dr. Charles Stanley McVicar of the Staff of The Mayo Clinic died suddenly at 4:00 p. m. June 29, at Winona. He had gone to Winona to play golf. At the second hole, he felt so ill that he returned to the club house, where he collapsed in the shower bath. The cause of death was coronary sclerosis.

Dr. McVicar was forty-nine years of age, was born April 5, 1880, in MacGillivray township, Middlesex County, Ontario. He was educated in the public and high schools in Ailsa Craig and Parkhill. At the age of nineteen he enlisted with the Canadian forces for service in South Africa, and served two years with the Strathcona Horse. On his return to Canada he entered the Western Medical College, London, where he studied for two years, from 1902 to 1904. The last two years of his medical course were taken at Toronto University, where he was graduated as Silver Medalist in 1907. He served eighteen months as intern at the Sick Children's Hospital, Toronto, and six months at the Manhattan Hospital, New York. On completion of his hospital training he engaged in practice in Toronto and was associated with the teaching staff of his Alma Mater.

During the World War Dr. McVicar again served with the Canadian Forces, this time in Salonika, where he was mentioned in dispatches for distinguished service, March 29, 1927, by General Milne. He was promoted to the rank of lieutenant colonel and served as Chief of Medicine in the Orpington General Hospital, England. From here he was recalled to Toronto to take charge of the Christie Street Orthopedic Hospital, which was the central orthopedic institution of the Canadian Army.

Dr. McVicar became a member of the medical staff of The Mayo Clinic in January, 1921. His interest was largely in the field of gastro-enterology. Owing to his outstanding ability, personality and industry he was soon made head of a section in gastro-enterology and Associate Professor of Medicine, The Mayo Foundation Graduate School of the University of Minnesota. He was deeply interested in teaching and investigation. In ward rounds he was usually attended by many visiting physicians, who quickly learned to recognize his ability as a teacher. He made several solid contributions to our knowledge of diseases of the gastro-intestinal tract and liver, in part due to his interest in the borderline between medicine and surgery in these diseases.

In 1912, he married Miss Mary Gillies of St. Marys, Ontario, who together with his son George and daughter Jeanette survives him. He is also survived by his mother, of Ailsa Craig, Ontario, and by his brother George of Cayley, Alberta. He was a member of the Presbyterian Church.

His broad viewpoint in medicine, his interest in both the clinical and scientific sides of medicine, his firm, sincere and fearless character, likable personality, dry sense of humor, his love of home and capacity for friendship marked him for leadership He was an excellent speaker and was in great demand for medical meetings throughout the United States and Canada. In the death of Dr. McVicar the Staff of The Mayo Clinic has lost one of its most valued and best loved members.

Louis McCargo Fowler

1901–1929

Dr. Louis McCargo Fowler, Fellow in Surgery in The Mayo Foundation since July 1, 1926, was drowned in Lake Pepin, near the Frontenac Inn, about 9:30 p. m. on Saturday, June 29, 1929. The day had been exceedingly warm, and just before nightfall a party set out from Rochester for a swim. Dr. Fowler started to swim to the raft alone in the darkness and apparently was seized with cramps. He called for help, but attempts at rescue by other members of the party, none of whom were strong swimmers, failed. The body was recovered about forty minutes after the accident. Unsuccessful attempts at resuscitation were made.

Dr. Fowler was born in Greensboro, North Carolina, September 27, 1901. He attended the University of North Carolina for four years and the University of Pennsylvania, where he received the degree of doctor of medicine in 1924. He served internship at the Robert Packer Hospital, Sayre, Pennsylvania. Dr. Fowler was well liked, and success was foreseen for him by his associates. At the time of his death, he was working in the Section on Orthopedics in the Division of Surgery, under the direction of Dr. M. S. Henderson.

LEVULOSE AND ARTICHOKES IN DIABETES

There is still considerable dispute as to the action of levulose in diabetes. One thing that stands out is that although levulose is well borne at first, hyperglycemia and glycosuria soon make their appearance. Jerusalem artichokes contain approximately 16 per cent of carbohydrate, chiefly inulin, which on hydrolysis yields levulose. Joslin showed that it was possible to substitute 15 Gm. of carbohydrate from Jerusalem artichokes for 5 Gm. from 5 per cent vegetables, but has cautioned that a small dosage should be used to avoid the danger of gastric upset. At a recent meeting of the American Chemical Society a paper was read reporting experiments in which varying quantities of dried artichoke and inulin were used in the diet of a diabetic patient: the conclusion reached was that Jerusalem artichokes are of doubtful value as a source of carbohydrate for diabetics. (Jour. A. M. A., June 22, 1929, p. 2121.)

A PAGE FORUM OF THE COMMITTEE ON PUBLIC HEALTH EDUCATION

Doctor Bills in Installments

Clipping from *St. Paul Pioneer Press:*

"As a counter move against socialized medicine, characterized by free clinics and publicly supported medical service, the Chicago Medical Society has organized a finance corporation through which doctor bills can be paid on the installment plan. Operating after the manner of companies which finance automobile or radio purchases, the credit corporation will pay physicians a part of their fee promptly, take 13 per cent of the total as its share, and finance the patient, charging him 6 per cent interest on the unpaid balance of bills.

"The society has high hopes that this plan will help combat some tendencies it has been attacking. Sponsors believe indiscriminate charity will be discouraged and that worthy persons will be able to engage competent medical care on payments commensurate with their ability to pay. A financial arrangement of considerable convenience to doctors and patients will be created. Physicians will be served by an efficient collecting agency and will be protected against losses which are one reason for high costs of medical care.

"Millions of people already pay doctor bills on the installment plan. But at present it is a hit-or-miss kind of method under which medical service is available regardless of credit ratings, by which generous doctors work hard for meager returns and crafty people get free attention that they could pay for if they wished. The Chicago Medical Society has only systematized and organized practices which have long existed."

Socialized Medicine

Clipping from *St. Paul Pioneer Press:*

"The danger of socialized or state medicine, which has agitated the medical profession throughout this country, has been made the subject of an address by Dr. Charles B. Reed on his inauguration as president of the Chicago Medical Society. In Chicago the issue has caused a sharp controversy in which the society itself has participated.

"The day when socialized medicine triumphs will not be a fortunate one for America. If treatment of human ailments is to become increasingly a function of the state, then indeed physicians and patients will encounter complications of their existing troubles.

"But the medical profession may be sure that state medicine will not be a causeless development. If it comes, it will be the result of some failure to meet human needs with existing methods. If the medical profession will examine the problem frankly, it probably will find that the demand for state or socialized medicine springs from popular fear of financial burdens of medical service. The public has high confidence in the modern doctor's skill. But when sickness comes, for large numbers of middle class families the choice now lies between deliberately neglecting personal health, acceptance of charity or the assumption of debts for expensive specialized service which may take years to pay.

"No propaganda nor flair for socialism in America, where reliance upon individual initiative is so prevalent, can usher in state medicine if adequate medical service is made accessible to the public at costs which are within average means."

OF GENERAL INTEREST

Dr. S. F. Herrmann, formerly of Rochester, Minnesota, has moved to Tacoma, Washington.

Dr. H. N. Klein of Saint Paul has returned from Vienna, Austria, where he has been attending medical clinics.

Dr. J. C. Litzenberg of Minneapolis is in Seattle, where he will deliver a course of lectures at the University of Washington.

Announcement has been made of the engagement of Florence Eugenie Watkins of Winona to Dr. Donald W. DeCarle of Rochester.

Dr. and Mrs. Angus Morrison of Minneapolis with their four children, Clinton, Angus E., Anne and John V. Morrison, have returned from a four months' trip abroad.

Dr. and Mrs. Archie H. Beard of Minneapolis have returned from a three months' trip abroad, visiting England, Scotland, Sweden, Denmark, Austria, Switzerland and France.

Dr. H. Longstreet Taylor of Saint Paul has been elected head of the Ramsey County Public Health Association for the coming year. Dr. Taylor succeeds Mr. Edward P. Davis.

Dr. Scott Searles of Minneapolis, who has practised medicine in Minnesota since June, 1889, has announced his retirement, and the sale of his radium and laboratory interests to Dr. Ignatius J. Murphy.

Dr. Alfred W. Adson of Rochester has been appointed to the state board of medical examiners for a period of seven years, his term expiring in May, 1936. Dr. Adson succeeds Dr. Leon A. Barney of Duluth.

Dr. and Mrs. Wallace H. Cole of Saint Paul are attending the eleventh conference of the International Orthopedic Association in London. They will travel in England and Scotland before returning home in September.

Notification of his promotion to a captaincy in the medical officers' reserve of the United States army has been received by Dr. Alfred B. Hart of Owatonna. Captain Hart had previously been commissioned as first lieutenant.

The wedding of Dr. E. C. Hansen, Fairview Hospital, Minneapolis, and Miss Cecilia G. Kaupang of Northfield, Minnesota, was solemnized Saturday, June 15, 1929. Dr. and Mrs. Hansen are now at home in Minneapolis.

Dr. Ruben Nomland, formerly of The Mayo Clinic, Rochester, Minnesota, has announced the opening of an office at 55 East Washington Street, Suite 3705 Pittsfield Building, Chicago, for the practice of dermatology and syphilology.

Both Dr. W. J. Mayo and Dr. Charles H. Mayo attended and took part in the meeting of the British Medical Association at Manchester, England, July 22 to 25, 1929. Both received the degree of Doctor of Laws from the University of Manchester during their visit.

Dr. J. T. Asbury of Rochester, Minnesota, was accidentally killed the morning of July 4 while driving a tractor on the grounds of his new home north of town. Dr. Asbury was formerly a member of the State Medical Association but had largely retired from the practice of medicine.

Dr. Walter H. Ude of Minneapolis has announced the purchase of the *X*-ray Department of the Minneapolis Clinical Association, 701 La Salle Building. The department will be operated in the same location and in the same manner as in the past, Dr. Leo. G. Rigler acting as consulting roentgenologist.

Dr. Charles W. Hughes, who has charge of clinical research at the Veterans Hospital No. 106, Fort Snelling, has been transferred to Chicago. The clinic at Fort Snelling, which has been rated as one of the best in the country, has been conducted under the direction of Dr. Hughes since its inception when the hospital was opened Jan. 1, 1927.

Dr. Howard McI. Morton of Minneapolis has been chosen as a lecturer at the thirteenth International Ophthalmological Congress to be held at The Hague, Holland. Dr. Morton left in July and will be gone about three months. The congress meets every five years and its delegates go from all parts of the world to attend. The last meeting, five years ago, was held in the United States.

The American Association for the Study of Goiter has offered a prize of three hundred dollars and a medal of honor for the author of the best essay based upon original research work on any phase of goiter. Manuscripts must be in the hands of the corresponding secretary, J. R. Young, Rose Dispensing Building, Terre Haute, Indiana, by July 4, 1930. The purpose of the offer is to stimulate research work on the subject of goiter.

Dr. B. T. Horton of Rochester has been advised that he has been awarded the John Horsley memorial prize of $1,000. This prize was founded in 1925 by Dr. J. Shelton Horsley, as a memorial to his father. The prize is awarded every two years by a committee of the Medical Faculty of the University of Virginia for the best thesis on some subject in general surgery. It is open to all graduates of the Medical Department of the University of Virginia of not more than fifteen years standing. The first award was made in 1927 to Dr. C. B. Morton, a fellow in surgery in The Mayo Foundation. The subject of Dr. Horton's thesis was, "A study of pyloric block with special reference to musculature myenteric plexus and lymphatics."

CONSULTATION BUREAU

WM. A. O'BRIEN, M.D., *Director*
Minnesota State Medical Association
11 West Summit Avenue
Saint Paul, Minnesota

1. *Question.*—Three year old girl fell and fractured the right femur immediately below the great trochanter. The fracture line is oblique, and the bones are in good position. She was treated in her home with vertical suspension and nine pounds of weight for traction to prevent overriding. Measurements showed about 1 cm. shortening at the time of injury, and at the present not over .5 cm. It is now four weeks after the accident, and I think the traction and suspension apparatus should be removed, and the patient allowed to lie in bed without any splint. Can she safely be allowed out of bed? What type of support will she need when she is up? How long should mechanical support be necessary? Is a walking type of Thomas splint the most satisfactory? Would a Thomas splint be adequate protection for the soft callus? Does such a splint have to be made for a patient? *X*-rays will be taken some time after traction is removed.

   Answer.—The following reply is based on the supposition that the fracture line is intertrochanteric, *i.e.*, one extending through the greater and lesser trochanters. This type of fracture when it heals gives better union than a similar fracture line in the shaft because of greater surface area of bone involved. Instead of taking the *x*-ray some time after the traction is removed, take your plates before any changes are made, as the future treatment of the case depends entirely on the amount of callus which is present. The patient should be kept in bed with a weight applied for at least five or six weeks instead of four. If this is done, it may not be necessary to use any other type of splint. When she is allowed out of bed, have her use crutches and at the end of seven or eight weeks allow her to walk alone. The *x*-ray should be your final guide before making any changes in the treatment.

2. *Question.*—Is it possible for me to run a drug store in connection with my practice? There is no other drug store for fifteen miles.

   Answer.—The State Board of Pharmacy informs us that no one except a registered pharmacist can run a drug store. It is possible for a physician to conduct a general store in connection with his practice and dispense his own medicines. It is not proper for him to call the place a drug store nor to fill other doctors' prescriptions unless a registered pharmacist is also employed.

3. *Question.*—Patient became ill about five weeks ago with cervical adenitis, followed in ten days by a swelling over the clavicular region. The swelling has now extended down on the left side to the mammary gland, and there is also marked edema of the left arm. Dyspnea suggests mediastinal pressure. The pulse is rapid, and the temperature is low. We drained the cervical region and found nothing. The fluid resembled lymph. Can this swelling be caused by an accumulation of lymphatic fluid, causing the pressure on the subclavian vessel? Examination of the gland by a pathologist showed no lymphosarcoma or lymphadenoma. We removed some milky fluid from the left chest. If this is a pleuritic exudate, I cannot understand the marked edema of the left arm.

   Answer.—The fluid from the chest of your patient is chyle. The probable source is obstruction with rupture or diapedesis of the thoracic duct or lymphangitis. The following conditions should be considered:

   1. Disease of the nodes (tuberculosis, Hodgkin's disease, lymphosarcoma, leukemia, suppurative adenitis)
   2. Aortic aneurysm
   3. Other mediastinal tumors (dermoid cyst, fibroma, metastatic carcinoma)
   4. Aberrant thyroid
   5. Thymoma
   6. Tumor of the esophagus (benign or malignant)
   7. Paravertebral abscess
   8. Diverticulum of the esophagus
   9. Encapsulated empyema

   The following procedures should be done: Fluroscopic examination, a flat plate of the chest, study of the mediastinum by the *x*-ray in the lateral view, blood examination, study of the contour of the barium-filled esophagus, and reexamination of the excised lymph node. If there is a mediastinal mass which does not pulsate and the blood shows no signs of leukemia, it would be advisable to give the patient the benefit of the doubt and give a deep *x*-ray treatment over the mediastinum to relieve pressure. If the tumor responded to this treatment, it would mean that it was probably Hodgkin's disease, lymphosarcoma, thymoma, or aleukemia.

4. *Question.*—We have a patient with an aortic aneurysm, who has been under anti-syphilitic treatment for three years. We have been using neosalvarsan, mercury rubs, and some potassium iodide. The blood Wassermann reaction remains positive, and we are wondering if you have any suggestions for more effective treatment.

   Answer.—If your patient has no other manifestations of syphilis, *e.g.*, neurosyphilis, and he is in good condition and has had no recent weight loss, the following treatment is advised: Give neosalvarsan at weekly intervals over a period of ten to twelve weeks. Three such courses should be given during the coming year. Very little success is obtained by using bismuth, mercury, or iodides, and these drugs are usually omitted in the treatment of syphilis of the aorta.

MISCELLANEOUS

SUMMARY OF THE NATIONAL TUBERCULOSIS ASSOCIATION MEETING, ATLANTIC CITY, MAY, 1929*

The Twenty-fifth Annual Meeting of the National Tuberculosis Association was a very interesting and instructive one. There were a number of papers presented showing new and valuable work done during the past year, the gist of which I wish to give you at this opportunity.

Dr. Eugene Opie, President of the Association and recipient of the Trudeau Medal for meritorious work during the past year, gave the opening address: The Epidemiology and Control of Tuberculosis. His paper concerned itself with facts relating to the wide dissemination of tuberculous infection, the uncertainty of its nature and the great need for more penetrating work amongst the children of grade school and high school age. A survey of high school children in Philadelphia showed that one in every hundred presumably healthy school children between the ages of 12 and 18 years has pulmonary tuberculosis, and that a larger number have a latent infection that has not as yet impaired health. Knowledge of the occurrence of tuberculous infection in the community and of the conditions that favor or retard its spread was considered the basis of all effective efforts to control the disease.

Dr. Walter Rathbun of Cassadaga, New York, also presented an interesting paper on juvenile tuberculosis. For the past six years, the Newton Memorial Hospital group has worked in the schools of Chautauqua County and the results were convincing that the public school is one of the most important fields in the control of tuberculosis. Definite evidence of latent tuberculosis was found in three and four per cent of the total school population. It was startling to learn that in the majority of high school students with pulmonary tuberculosis no history of reliable symptoms could be obtained either from the students themselves or the parents. Their disease, which was unmistakably evidenced and usually past the minimal stage, was discovered on routine *x*-ray and physical examination. Another interesting and valuable observation made was that in going into the home and *x*-raying the family group, the source of infection of these students in most instances was found.

Dr. H. H. Hetherington of Philadelphia gave some more instructive information relative to the occurrence of tuberculosis among the juvenile population of the public schools. Of 1,425 children between the ages of 15 and 19 years, 1.5% were found to have apical tuberculosis. Fifteen per cent had demonstrable tuberculosis of the tracheo-bronchial lymph nodes.

The work of Drs. Fenger and Mattill of Glen Lake Sanatorium proved to be a very interesting study of 3,011 school children in Hennepin County.

The impression gained from the papers on juvenile tuberculosis was the urgent need for thorough tuberculosis surveys in our public schools. The surveys could not possibly be complete or effective without the diagnostic use of tuberculin and the *x*-ray, for it was with the use of these measures that the most reliable information was gained. I was struck by the thought that we have in the State of Minnesota a splendid opportunity to contribute materially to the work already being done in this field and to gain an intelligent understanding of the actual amount of infection and disease present in the various communities. As Doctor Opie stated, such information is the basis for the gaining of effective control over this widespread disease.

The research side of tuberculosis showed the development of some very interesting work. Dr. S. A. Petroff of Saranac Lake, New York, presented his studies, revolutionary in nature, on "Microbic Dissociation of the Tubercle Bacilli." After twenty years of patient research work in the tuberculosis laboratory, he had finally accomplished a work which gave him satisfaction and held forth great promises. He found that if a typical virulent tubercle bacillus is cultivated on artificial culture media, two strains develop from it: the R strain and the S strain, indicating resistent to environment and sensitive to environment. The R type is the variant from the parent organism and is not very pathogenic, is stable, and does not produce symptoms. After a drop in temperature of the patient to normal and a loss of symptoms, there is found to be a predominance of the R type of organism. The S type is virulent, producing symptoms, and predominates at the time when fever is present. Reversibility of R into S may take place not only in the human and animal body, but by special cultural methods. Because of this possibility, Dr. Petroff is opposed to the Calmette vaccination method of children. There is no guarantee that an avirulent organism will not revert to a virulent one. Dr. Petroff is still in the midst of research work with these organisms, and when it is completed we can expect some very illuminating information on the subject of lesions and symptoms.

The very commendable report of Drs. Sabin, Doan and Forkner of the Rockefeller Institute showed the reaction of the tissues to the protein and lipoid fractions from the tubercle bacilli. A considerable amount of work had been done by injection of the various fractions singly into animals and noting the reaction to each individual product. Briefly stated, it was found that the toxic symptoms of the disease could be produced by the protein material and the specific lesions by the lipoids.

The research work of Dr. Leroy Gardner of Saranac lake on dust inhalation indicated that dust material such as quartz and to a lesser extent carborundum and granite would reactivate healing foci of primary infection.

One of the most valuable and encouraging papers of the meeting was the report made by Drs. David Smith and M. McConkey, Ray Brook, New York, on "Experimental Tuberculosis in the Guinea Pig Induced by

*This report of some of the important papers presented before the National Tuberculosis Association is a reprint of a letter sent to sanatorium superintendents of the state.

Feeding Tubercle Bacilli to Animals Living on a Diet Deficient in Vitamins." The special diet consisted of:

White bread, 20 per cent
Chipped beef, 20 per cent
Potatoes, 20 per cent
Oatmeal, 15 per cent
Skimmed milk, 15 per cent
Sugar, 10 per cent

It is adequate in calories, in protein of good quality, in mineral salts and in Vitamin B. It is relatively deficient in Vitamins A, C, D. It was found that guinea pigs fed on this vitamin-deficient diet and also given tubercle bacilli by mouth developed intestinal tuberculosis. Those fed on the same diet, but supplemented by 0.3 c.c. of cod liver oil and 10 c.c. of tomato juice daily, were protected from the development of this complication. Their work pointed clearly to the fact that a vitamin-deficient diet so alters the resistance of tissue as to make it vulnerable to tuberculous disease.

The results of the clinical application of cod liver oil and tomato juice therapy to tuberculous enteritis cases was presented by Dr. McConkey. Statistics, as well as careful clinical observations on 110 patients, showed the unquestionable value of this form of treatment for intestinal tuberculosis. It is administered as follows: one-half ounce of cod liver oil is floated on the surface of three ounces of ordinary tomato juice. The whole is chilled and served immediately after meals. It must be served ice cold. The work as presented gave one this impression. Here was a very simple and economical form of therapy which was valuable not only in the treatment of intestinal tuberculosis, but also for its prevention. If a properly balanced vitamin diet is effective in lesions along the intestinal tract, it would appear that it should also benefit tuberculous lesions elsewhere. The facts as presented, relating to this simple therapy, left me with the conviction that it should be given the benefit of a trial in all our sanatoria. It should be continued over a long period of time and careful observations made in an attempt to gain still more scientific data concerning its use and its results.

We have all wondered as to the length of time necessary to keep a lung collapsed in order to insure against a relapse. This subject was ably dealt with by Drs. J. Burns Amberson, Jr., and H. McL. Riggins, Loomis, New York. A paper was presented entitled, "A Report on One Hundred Sixty-seven Tuberculous Patients with Lungs Re-expanded After Artificial Pneumothorax Treatment." From their extensive experience they concluded that to insure the best ultimate results the pneumothorax treatments should be continued over a period of two to three years at the minimum. The 167 cases which they followed were patients where re-expansion of the lung had been present for five or more years. The best results were obtained in that group which showed a good collapse with closed cavities and over a three year period of collapse. Eighty-four per cent of these were living and 72 per cent were well. When one considers that these cases were selected from patients five to fifteen years after re-expansion had occurred, it gives us a rather encouraging picture of the ultimate fate of the pneumothorax patient. When one further considers that pneumothorax cases are selected from that group of patients who are not responding to ordinary bed rest and who otherwise would proceed to a fatal termination, then we are struck by the importance of this therapeutic weapon. From the work presented at this meeting, the impression was gained that collapse therapy has won a permanent place in the treatment of pulmonary tuberculosis.

I have included herein only those facts that I considered the most interesting and instructive. Time will not permit a résumé of the other splendid papers presented, but which you will be able to peruse when the proceedings of the national meeting are made available.

There is another element of importance relating to such gatherings that must be mentioned and that is the valuable contacts made with workers in various fields and in different localities. There can be no doubt but that the stimulus so gained is something which we should all attempt to make available for ourselves. It reflects itself in a favorable way upon our endeavors to combat successfully the tuberculosis problem.

Yours very sincerely,
ARNOLD S. ANDERSON, M.D.
Executive Secretary.

OXYGEN IN THE TREATMENT OF CORONARY OCCLUSION

PRELIMINARY REPORT

ROBERT I. RIZER, M.D.
Minneapolis

The results which I have gained from the use of oxygen in cases of coronary occlusion have been so good that I wish to make a preliminary report of its use in my cases. In my experience, previous to the use of oxygen, no treatment gave immediate relief of symptoms in an acute attack. This was due in part to the fact that the cause of the pain was not understood. An article by Kiefer and Resnick suggested that anginas were possibly due to anoxemia of the heart muscle. Acting on that suggestion, I determined to try oxygen in my cases of severe anginas and coronary occlusions. The results were that the patients have been relieved of symptoms which had not yielded to any of the usual forms of treatment. The relief of symptoms in all cases where oxygen was used came within two minutes after the application of the oxygen tent.

The relief of pain following the use of oxygen suggests that the pain in the attack is due not to a spasm of the vessels but to anoxemia of the heart muscle.

In the cases treated, the patient was placed in bed with the head slightly elevated. The oxygen tent was applied, and the apparatus adjusted to maintain a 50 per cent oxygen atmosphere. This treatment was continued from twelve to fourteen hours. The tent was

then removed, and the usual cardiac supportive measures were used.

The cases treated will be reported in full in a subsequent paper.

1931 Medical Arts Building.

OUR COUNTRY DOCTOR

We sing the praise of authors, of warriors and of men,
Who've climbed to heights of glory by their deeds of sword or pen;
We praise the bard, the statesman, the sportsman and the man
Who holds the fate of nations in the hollow of his hand.

But there's perhaps another who has ne'er received his due,
Who's spent long years of service in his work for me and you;
And though his deeds may never be proclaimed from shore to shore,
In fulfilling Christ's own mission other men have done no more.

No midnight call that came to him e'er fell on deafened ear,
The steady voice that answered seemed to drive away all fear;
No night too wild, no road too long, naught seemed to bar his way,
His aim was "Render service without one thought of pay."

For fifty years he's labored in a field of pain and strife,
As he traveled from youth's morning to the eventide of life;
He has eased the pain of thousands, he's rejoiced with us when glad,
And when death came to our household his heart too was sick and sad.

And we folks who do so little in behalf of humankind,
If we'd view his service record, one great lesson we might find;
The man who's in the limelight truly may be great and fine,
But we'll match our country doctor's with his record anytime.

MRS. ANNA FARRELL,
Farmers' Elevator Guide, July, 1929.

REPORTS AND ANNOUNCEMENTS OF SOCIETIES

THE AMERICAN PUBLIC HEALTH ASSOCIATION, THE AMERICAN CHILD HEATH ASSOCIATION

September 30 to October 5, 1929

The arrangements for the meetings of the American Public Health Association, the American Child Health Association and their related organizations in Minneapolis listed in the July number of MINNESOTA MEDICINE, are well under way. The preliminary program of the visiting health associations appears in the July number of the American Journal of Public Health.

The Conference of State Sanitary Engineers will begin its sessions at the Curtis Hotel, Minneapolis, on Saturday, September 28. The other members of the group, including the Conference of State Laboratory Directors, recently added to the promised gathering, will convene on Monday, September 30, at the Municipal Auditorium. It is hoped registration, which will begin Monday morning, will be practically completed on this date. Registration booths will be on the lower exhibition floor, adjoining the Commercial Exhibits. The headquarters of the American Public Health Association will be found at the southeast corner of the same area.

THE NORTHWEST CONFERENCE FOR CHILD HEALTH AND PARENT EDUCATION

The third annual session of the Northwest Conference for Child Health and Parent Education will open at the Auditorium on Wednesday, October 2, at 9:00 a. m. Its registration office will be on the first or street-level floor, near the Northwest entrance (Grant Street and Stevens Avenue). The ticket sales for the Conference have been abandoned for this year. Instead, registration is required, under a registration fee of One Dollar. Upon its completion, which can be secured now, and by mail, tickets of free admission will be issued which will cover attendance upon ALL meetings of the Conference, upon ALL sessions of the American Public Health Association, the American Child Health Association, and the related organizations as listed, and upon the Educational Health Exhibit.

The meetings of the Northwest Conference for Child Health and Parent Education will cover one and a half days, the morning sessions of Wednesday, October 2, and of Thursday, October 3, and the evening session of Wednesday, October 2, held conjointly with the American Child Health Association, occurring in the main audience room of the Auditorium; and the

luncheon and afternoon sessions of Wednesday, October 2, at the Curtis Hotel.

The adjournment of the Northwest Conference at noon on Wednesday is planned to give the members a special opportunity to visit and study the Educational Health Exhibit on the afternoon and evening of that day.

A preliminary program of the Northwest Conference will appear shortly.

A Popular Health Program, consisting of health talks, demonstrations, radio, delineascope and lantern slide exhibit is planned on the afternoon of each day from October 1 to October 5. Some excellent films will be presented.

Members of state organizations meeting conjointly with the national groups are urged to become members of the latter. If they do not desire to do this, they will be eligible to registration under The Northwest Conference for Child Health and Parent Education.

VISITORS TO THE MEETINGS

All visitors to the health meetings, who are not registered members or fellows of the national organizations or registered attendants upon the Northwest Conference for Child Health and Parent Education, will be asked to register as Visitors under a registration fee of One Dollar. Registration for visitors will open, coincidently with that for other groups, on Monday, September 30, at 9:00 a. m.

The Educational Health Exhibit, presented under the joint auspices of the American Public Health Association and the Northwest Conference for Child Health and Parent Education for the benefit of the people at large, will open on the morning of Tuesday, October 1, and will close at noon of Saturday, October 5. Admission will be by ticket, but without entering charge.

A large number of organizations are contributing to the creation of the educational exhibit. It will be housed on the first or street-level floor. It will interpret health to the people in very broad terms. It will be arranged under divisions of sanitation, or community hygiene; of disease discovery and prevention; of child development; of recreational activities; and occupational activities.

Attendants, in charge of each booth or group of booths, will reinforce the visitors' direct observation by any information they may seek.

The time has surely come when health education must be carried directly to the people and in plain, unmistakable terms. There is nothing concerning which the public is more clearly entitled to know. Those of the seeing eye and the hearing ear and the comprehending mind will find the opportunity in this exhibit to learn much of living interest and saving knowledge.

RICHARD OLDING BEARD, M.D.
Executive Secretary.

NORTHERN MINNESOTA MEDICAL ASSOCIATION

The annual assembly of the Northern Minnesota Medical Association will be held at the Hotel Birchmont, on Lake Bemidji, at Bemidji, Minnesota, Monday and Tuesday, September 2 and 3, 1929. A two day program, with scientific papers by leading men in the state and visiting guests, has been planned. Entertainment will also be provided for the ladies. This is the annual outing meeting of the Association and a large attendance is expected.

The program for the two days will be as follows:

PROGRAM

Monday, September 2

9:30 A. M.–11:30 A. M.

MARY GHOSTLEY, M.D., International Falls
"Eclampsia and Its Mortality"

RALPH E. WEIBLE, M.D., Fargo, North Dakota
"Peptic Ulcer"

F. WHITMORE, M.D., St. Paul
"Psychoanalytic Problems"

A. M. SNELL, M.D., Rochester
"Some Recent Advance in Endocrinology"

M. G. GILLESPIE, M.D., Duluth
"A Study of Postoperative Hypothyroidism"

Luncheon, 12:00 A. M.–1:30 P. M.

N. O. PEARCE, M.D., Minneapolis
Subject to be announced

J. T. CHRISTISON, M.D., St. Paul
Subject to be announced

Pathological Symposium
W. A. O'BRIEN, M.D., Minneapolis
GEO. E. FAHR, M.D., Minneapolis
O. H. WANGENSTEEN, M.D., Minneapolis

Recess 3:00 P. M.–3:15 P. M.

L. L. BROWN, M.D., Crookston
"Immunity"

HENDRIE GRANT, M.D., St. Paul
"Eye Injuries"

W. F. BRAASCH, M.D., Rochester
"General Symptoms Secondary to Urinary Retention"

S. H. BOYER, M.D., Duluth
"Jaundice Problems"

Banquet 6:30 P. M.

Rev. W. C. SAINSBURY, St. Paul
M. O. OPPEGAARD, M.D., Crookston

Tuesday, September 3

8:30 A. M.–12:00 A. M.

A. L. MACDONALD, M.D., Duluth
Diagnosis and Treatment of Uterine Bleeding

MAX ALBERTS, M.D., St. Paul
Hyperthyroidism in Children

D. GRETH GARDINER, M.D., St. Paul
Bronchial and Esophageal Foreign Bodies

Business Meeting 10:00 A. M.

Gastro-intestinal Clinic
C. B. WRIGHT, M.D., Minneapolis
LEO RIGLER, M.D., Minneapolis
Diagnostic Dialogue

CAMP RELEASE DISTRICT MEDICAL SOCIETY

Camp Release District Medical Society met in a midsummer session in Ramsey State Park at Redwood Falls, Minn., July 1, 1929, at 2 p. m., with President A. A. Passer presiding.

Inasmuch as the University extension course had so recently been concluded it was decided to make the meeting a social as well as a profitable one.

Dr. J. T. Ohlmacher, Professor of Bacteriology and Pathology, Medical School of University of South Dakota, Vermilion, gave an excellent address on the subject of "Preventive Medicine in the Light of Modern Scientific Knowledge."

Dr. C. M. Anderson, associate in the department of Ear, Nose and Throat of The Mayo Clinic, presented his subject, "Sinusitis in Relation to General Medicine," in a very illuminating as well as entertaining manner.

After the discussions, golf and other entertainment was indulged in, following which a dinner was served by the ladies' auxiliary to sixty guests, including the doctors and their families.

Drs. H. B. Cole, W. A. Brand, Thomas Flinn, and Brainerd Flinn, all of Redwood Falls, were the entertaining hosts at this delightful meeting.

L. J. HOLMBERG, M.D.,
Secretary.

SOUTHERN MINNESOTA MEDICAL ASSOCIATION

The Southern Minnesota Medical Association met at Winona, June 15, in the Masonic Temple for a morning of Clinics. These included The Anemias by Dr. Frank Heck and Dr. C. H. Watkins of Rochester, Pediatric Clinic by Dr. J. C. Christison of St. Paul, a Neurologic Clinic by E. M. Hammes of St. Paul, and a Surgical Clinic by Dr. Owen Wangensteen of the University of Minnesota.

At noon the Society with their families and friends boarded the excursion steamer, Capitol, and lunch was served to 367 on the boat. While the guests and relatives enjoyed the Mississippi river scenery the members conducted a scientific session in an enclosed room on the mid-deck. It was interesting to note that practically every physician stayed throughout the meeting. The program was very interesting and well received. The program included, The Injection Treatment of Varicose Veins, by Dr. Lichtenstein of Winona; Peroral Endoscopy, by Dr. J. T. Schlesselman of Mankato; The Use of Iodized Oil in the Diagnosis of Thoracic Conditions, by Dr. E. K. Geer of St. Paul; Cases of Coronary Infarction with Survival, by Dr. J. A. Evans of La Crosse, Wisconsin; Recent Medical Development in the Treatment of Arthritis, by Dr. L. G. Rowntree of Rochester; Orthopedic Surgery in Arthritis, by Dr. Henry Meyerding of Rochester, and Bacteriologic Studies in Arthritis and Endocarditis, by Dr. B. J. Clawson of Minneapolis.

The evening session followed a banquet in the Masonic Temple and included the business meeting, a talk by Dr. W. J. Mayo and a talk by Mr. Cox on Upper Mississippi Wild Game and Fish Refuge. This was followed by several reels of motion pictures of African Big Game Hunting, by Dr. A. R. Metz, Chief Surgeon of the Chicago, Milwaukee, St. Paul and Pacific Railroad. Officers elected were Doctor Waltman Walters of Rochester, president; Dr. J. T. Schlesselman of Mankato, first vice president; Dr. C. B. McKaig of Pine Island, second vice president; and M. C. Piper (re-elected), secretary-treasurer.

The society voted to offer a suitable prize for the best scientific exhibition at the State Medical Association Meeting or to the Senior Medical Student at the University of Minnesota who contributes the best piece of original work in his senior year. The next meeting will be held at Mankato in the fall of 1930.

M. C. PIPER, M.D.,
Secretary.

WABASHA COUNTY MEDICAL SOCIETY

The sixty-first annual meeting of the Wabasha County Medical Society was held at Wabasha, Minnesota, Thursday, July 11, 1929.

At the business session in the forenoon, the following officers were elected for the coming year: President, Dr. Russell H. Frost, Wabasha; Vice President, Dr. J. S. Collins, Wabasha; Secretary-Treasurer, Dr. W. F. Wilson, Lake City; Delegate to State Association, Dr. W. H. Replogle, Wabasha; Alternate, Dr. H. E. Bowers, Lake City; Censor for three years, Dr. J. F. Bond, Wabasha; Censors whose terms are unexpired are Dr. W. J. Cochrane, Lake City, and Dr. J. A. Slocumb, Plainview.

Topics pertaining to medical economics, welfare of the Society and public health education were discussed.

Dinner was served at Buena Vista Sanatorium, through the courtesy of the Sanatorium officials, to thirty-nine guests, including regular and affiliate members, dentists, visiting physicians, lay members of the Sanatorium Commission, representative of the press, and ladies accompanying.

The afternoon session was held on board the Yacht "North Star" tendered for the occasion through the courtesy of Doctor and Mrs. William J. Mayo.

During the boat ride on the Mississippi, the following program was presented:

President's Address—"Let Us Try," Dr. D. P. Dempsey, Kellogg.

"Careful Obstetrics," Dr. J. H. Simons, Minneapolis, Assistant Professor of Obstetrics and Gynecology, University of Minnesota.

"The Incidence of Tuberculous Infection in School Children," Dr. Russell H. Frost, Superintendent and Medical Director Buena Vista Sanatorium, Wabasha.

"Diagnosis and Treatment of Renal and Ureteral Calculi" (Lantern Demonstration), Dr. Philip F. Donohue, St. Paul. Department of Urology, University of Minnesota.

Appreciation of the entertainment features, and satisfaction with the program generally was individually spoken, and publicly expressed through a vote of thanks extended to all taking part in making this meeting a pleasant and instructive event.

W. F. WILSON, M.D.,
Secretary.

PROGRESS

Abstracts to be submitted to Section Supervisors.

Members are urged to abstract valuable articles which they run across in their reading and send the abstracts to the physicians in charge of the respective sections. In order to avoid duplication it would be well to communicate with one of the section supervisors before the article is abstracted.

PEDIATRICS

SUPERVISORS:

CHESTER A. STEWART,
LA SALLE BLDG., MINNEAPOLIS

ROY N. ANDREWS,
MANKATO CLINIC, MANKATO

CLINICAL SIGNIFICANCE OF THE WATER OF METABOLISM IN CHILDREN: Hyman Goldstein, M.D., New York (Arch. of Ped., June, 1929, Vol. XLVI, No. 6). The human body, particularly that of the child, is a huge chemical laboratory with an important irrigation system. Every kilogram of body weight contains from 0.6 to 0.7 kilogram water. A child weighing 50 pounds actually weighs 15 to 20 pounds in solids and 30 to 35 pounds in water content. Metabolism is a part of these chemical processes constantly going on in the body cells and fluids by virtue of oxidation. Water, as an intermediary in all these reactions, is absolutely necessary. The blood is being continually depleted of water and the loss must be made up by the ingestion of new water.

It is very essential that the child during health and disease keep a proper water balance. Rubner early called attention to the fact that in starvation an animal can lose most of its glycogen, fat and half of its body protein, approximately 40 per cent of this weight, and still live, whereas the loss of 10 per cent of water content of the body results in serious disorders, and the loss of 20 per cent results in death. Water is essential to the life and function of every living cell.

Diet modifies water balance a great deal. Carbohydrates and proteins favor water retention, but in excess, especially if protein is over 20 per cent of the total calories, metabolism and water loss is stimulated and hence weight loss. If carbohydrates are down to less than 3.5 per cent in the diet, acidosis may result. High fat diet encourages water loss and indirectly weight loss. In severe cases of vomiting with great loss of water and hydrochloric acid, there may be retention of alkalies, and alkalosis. There is no question but that marked changes in the water balance causes the same influence to partly change the alkaline reserve. Fortunately the latter content of the plasma is easily compensated and therefore more or less constant.

In considering the importance of keeping the water balance we must consider cause and effect in our clinical observations of diseases of children. We must recognize early symptoms and note the connection that exists between clinical states. This knowledge and clinical observation should give us intuition as to how and when to employ water therapy when indicated to prevent or treat dehydration and other serious conditions.

R. N. ANDREWS, M.D.

RESPIRATORY DISORDERS IN INFANTS: Harold Abramson, M.D., and Louis H. Barenberg, M.D., New York (Jour. A. M. A., June 29, 1929, Vol. 92, No. 26). An attempt was made to lessen the incidence of these infections by putting into practice the principles of "aseptic nursing." During the course of a winter all persons coming in contact with the infants in one ward were required to wear surgical masks and to scrub their hands thoroughly before the examination or the handling of the children. A comparison made with infants in a control ward where no such measures were carried out showed that a decrease in respiratory diseases of only 10 per cent was accomplished by this means. The greatest difficulties lay in occasional infractions of technic on the part of the nurses, as well as in the impossibility of absolutely preventing contact infections, from child to child. Infants who were kept in cribs showed almost a 50 per cent higher incidence of respiratory disorders than those cared for in bassinets. The frequency of infection per child was less in the protected than in the control ward.

Aseptic nursing was of no value to preventing the spread of an outbreak of varicella.

R. N. ANDREWS, M.D.

HYPEREMESIS HIEMIS OR THE WINTER VOMITING DISEASE: John Zahorsky, M.D., St. Louis (Arch. of Ped., June, 1929, Vol. XLVI, No. 6). Vomiting and diarrhea are frequent symptoms of respiratory infections. The disease usually begins suddenly, but often the older child complains of abdominal pains for one or two days preceding the acute attack.

The first alarming symptom is vomiting. This is violent and forcible and often accompanied by symptoms of collapse, *i.e.*, extreme pallor and prostration. The vomiting continues for one or more days.

Within a few hours after the vomiting begins, a looseness of the bowels is observed. Only exceptionally

have cells been found in the stools while in summer diarrheas a massive cellular content is generally present.

The young child lies for one to three days listless, sometimes in a semicomatose or stuporous state. The temperature in uncomplicated cases is not high. Recovery occurs invariably after a few days. The physical examination shows nothing of importance. There is no tenderness of the abdomen and only exceptionally is distention present.

R. N. ANDREWS, M.D.

APPENDICITIS IN CHILDREN: Charles E. Farr, M.D., New York (Arch. of Ped., June, 1929, Vol. XLVI, No. 6). It has been for years the author's practice, in hospital work, to consider every doubtful abdominal case as one of appendicitis.

If one errs on the side of being too radical, very little harm ensues. The operation is practically without danger; moreover a very probable source of future danger has been removed.

Tenderness, sensitiveness rather, at the usual site of the appendix, or along the outer border of the colon, or in the right side of the pelvis, by rectal examination, is highly suggestive.

It is only in the advanced stage of spreading peritonitis, or the overwhelming insult of an acute perforation, that we meet with the higher degrees of abdominal rigidity. Generally infants and young children will show more rise in temperature than similar cases in adults. In leukocytosis and polynucleosis the same is true. In *x*-ray if the appendix fails to appear at all on the plate, or if, having been filled, it fails to empty in 72 hours, or if, being surely located, it remains fixed in position and is tender on pressure, the diagnosis is almost certain.

Survey of a series of cases (1,459 in number) of appendicitis in children seems to prove:

1. That appendicitis is very common in childhood.
2. Chronic appendicitis, or a crippled appendix may exist from birth.
3. The difficulty of diagnosis is great but not insurmountable.
4. Chronic appendicitis shows practically no operative mortality in childhood.
5. Early diagnosis of acute cases and early operation yield just as good results in children as in adults.

R. N. ANDREWS, M.D.

THE NINHYDRIN TEST IN PREGNANCY

The Abderhalden Ninhydrin test for pregnancy has fallen into disrepute. There is no evidence that a specific ferment exists in pregnancy. While tests on serum from pregnant women are uniformly positive, the large number of positive results on the serum of men and non-pregnant women proved the test of no value for the diagnosis of pregnancy. (Jour. A. M. A., March 9, 1929, p. 829.)

EYE, EAR, NOSE AND THROAT

SUPERVISORS:

VIRGIL J. SCHWARTZ,
PHYS. & SURG. BLDG., MINNEAPOLIS

ARTHUR C. DEAN
CROOKSTON CLINIC, CROOKSTON

A STUDY OF POST-TONSILLECTOMIZED INDIVIDUALS: Arthur J. Wagers, M.D., Philadelphia, Pa. (The Laryngoscope, May, 1929, XXXIX, 310). The decision to remove the tonsils in the patients coming to the clinic of a large hospital is often based upon the most superficial examination and study of the patient.

Tonsils which have once been attacked by acute inflammation of the follicles are prone to subsequent attacks, so that in time the tonsil tissue shows evidence of chronic infection, and this local condition, resulting in absorption of toxins, is, in large measure, responsible for frequent attacks of various ailments in the case of school children. Chronic otorrhea and impairment of hearing are conditions which, to my mind, are only very indirectly affected by the presence of infected tonsils. In some instances their removal may be helpful in restoring the ears to normal, but I believe it is a mistake to tell patients nothing can be done for their ears until the tonsils have been removed.

The cases in this particular series number 274 of all ages, from two to sixty years. They have been divided arbitrarily into three age groups—those under eleven years, those from eleven to twenty-five, and those above twenty-five years of age.

Twelve months after operation, from the first group we had forty-six reports of conditions present. Twenty had been free from sickness and twenty-six reported disturbances of minor importance. In the second group twenty-three reported. Twelve of these had not been ill since the operation, eleven had been ill at some time and from more serious complaints than those suffered by the younger group. In the third group we had fifteen re-examinations out of an original forty-one. There had been no postoperative illness in five of these, while ten had suffered from various ailments.

It is noted with some satisfaction that the majority were benefited, but this study also serves to impress on us the fact that the tonsils are not alone responsible for all the suffering to which human flesh is heir.

CONCLUDING SUMMARY

Original examinations were made of 274 cases.

Subsequent interviews or reports were obtained in 84 cases.

No illness within one year or longer following operation reported by 37.

Illness of some description reported by 47. Definitely improved by operation, 63. Not improved by operation, 21.

ARTHUR C. DEAN, M.D., F.A.C.S.

ROENTGENOLOGY

SUPERVISORS

LEO G. RIGLER
MINNEAPOLIS GENERAL HOSPITAL, MINNEAPOLIS

A. U. DESJARDINS
MAYO CLINIC, ROCHESTER

CHOLECYSTOGRAPHY: Byrl R. Kirklin, M.D. (Arch. of Surg., June, 1929, Vol. 18, No. 6. Pp. 2246-2256). The advances in roentgenologic diagnosis of disease of the gallbladder have been rapid since 1924, in which year Graham and Cole announced calcium tetrabromphenolphthalein as a cholecystographic agent. The questions of the advantages of various dyes, the use of the oral or intravenous method of administration, the real place of cholecystography in medicine are subjects which had occupied considerable space in the literature.

Doctor Kirklin reviews the subject and analyzes the present status of this means of diagnosis beautifully.

At the Mayo Clinic sodium tetraiodophenolphthalein has been given orally to many thousand cases without untoward results and the method compares favorably with results obtained by intravenous checks on selected cases.

Concerning efficiency he brings out four main points which account for error in diagnosis and the variance in results in different sections of the country.

1. Lack of knowledge as to the line of demarcation between the normal and abnormal gallbladder from the surgeon's, anatomist's, physiologist's and histopathologist's standpoint.

2. Differing conceptions as to the cholecystographically normal and abnormal gallbladder.

3. The use or non-use of the clinical data in arriving at cholecystographic diagnosis.

4. Differences in the character of the material on which the computations are based and in the methods of analysis.

The author feels that a true roentgenologic diagnosis should be made without knowledge of the clinical history of the case; the results given to the clinician to whom properly belongs the correlation of all facts from all sources.

Reliable indicia of gallbladder abnormality are three in number:

1. Absence of shadow.
2. Faintness of shadow.
3. Mottling of the shadow.

Irregularities of contour seldom denote intrinsic disease.

Since cholecystography is preëminently a test of the ability of the gallbladder to receive and concentrate dye-laden bile, and since this capacity does not have a constant relation to health and disease, the report to the clinician should be in terms of function, not of anatomic condition. At the Mayo Clinic reports are given in three forms:

1. Normally functioning gallbladder.
2. Poorly functioning gallbladder, when the shadow is faint.
3. Nonfunctioning gallbladder when the shadow or viscus is not seen.

Evidence of stones may be reported in any of the above types.

Accuracy in diagnosis of cholecystic disease at the Clinic increased from 79 per cent in 1925 to 90 per cent in 1927, and has been on the increase since. This compares favorably with statistical reports.

In conclusion the author states that cholecystography is a remarkably able coadjutor, but, whatever it may reveal, the clinician cannot forsake his prerogative and duty to correlate the data with his own information, weigh the probabilities of disease, estimate its gravity and recommend appropriate treatment.

CHARLES MAYO, II.

NEW AND NON-OFFICIAL REMEDIES

The following article has been accepted by the Council on Pharmacy and Chemistry:

PARKE, DAVIS & Co.

Ampoules of Pitocin
Ampoules of Pitressin

TRUTH ABOUT MEDICINES

Insulin-Squibb, 80 units, 10 c.c.—Each c.c. contains insulin-Squibb (New and Non-official Remedies, 1929, p. 197) 80 units. E. R. Squibb & Sons, New York.

Diphtheria Toxoid.—A diphtheria toxoid (New and Non-official Remedies, 1929, p. 368) prepared from diphtheria toxin of which the L+ dose is 0.25 c.c. The toxin is treated with formaldehyde according to the specifications of the U. S. Public Health Service until it is detoxified. It is tested for antigenic power by subcutaneous injection into guinea-pigs. Diphtheria toxoid-P. D. & Co. is marketed in packages containing one bulb (0.5 c.c.) of dilute diphtheria toxoid for the reaction test and two bulbs (0.5 and 1.0 c.c. respectively) of diphtheria toxoid; also marketed in hospital packages. Parke, Davis & Co., Detroit.

Petrolagar (with Milk of Magnesia).—Liquid petrolatum (New and Non-official Remedies, 1929, p. 228) 65 c.c.; magnesia magma, 8 c.c.; emulsified with agar in a menstruum containing sugar, flavoring, sodium benzoate 0.1 Gm., and water to make 100 c.c. Petrolagar Laboratories, Inc., Chicago. (Jour. A. M. A., June 1, 1929, p. 1837.)

Bismarsen — Sulpharsphenamine Bismuth — Bismuth Arsphenamine Sulphonate.—The sodium salt of a bis-

muth derivative of arsphenamine methylene sulphonic acid with inorganic salts. It contains approximately 13 per cent of arsenic and 24 per cent of bismuth. Bismarsen is used in the treatment of syphilis. The drug is reported to be somewhat slower in its action than intramuscularly administered sulpharsphenamine or intravenously administered neoarsphenamine, but much more rapid than bismuth. More or less severe pains at the site of injection have been reported. Bismarsen is administered intramuscularly. Abbott Laboratories, North Chicago, Ill. (Jour. A. M. A., June 8, 1929, p. 1928.)

Digifoline-Ciba.—A digitalis preparation containing the active glucosides of digitalis, free from extractive matter. It is standardized to have the strength of digitalis leaves as standardized by the frog method of Focke. The actions and uses of Digifoline-Ciba are the same as that of digitalis. It may be administered orally, rectally, or by subcutaneous, intramuscular or intravenous injection. Digifoline-Ciba is marketed in the form of Ampules Digifoline-Ciba Solution, Digifoline-Ciba Liquid and Tablets Digifoline-Ciba. Ciba Company, Inc., New York.

Concentrated Pollen Extracts-Swan-Myers.—In addition to the products listed in New and Non-official Remedies, 1929, p. 26, the following product has been accepted: Canada Blue Grass Concentrated Pollen Extract-Swan-Myers. Swan-Myers Co., Indianapolis.

Sulpharsphenamine-Searle, 0.1 Gm. Ampules.—Each ampule contains sulpharsphenamine-Searle (THE JOURNAL, April 20, 1929, p. 1349) 0.1 Gm. G. D. Searle & Co., Chicago.

Sulpharsphenamine-Searle, 0.2 Gm. Ampules.—Each ampule contains sulpharsphenamine-Searle (THE JOURNAL, April 20, 1929, p. 1349) 0.2 Gm. G. D. Searle & Co., Chicago.

Sulpharsphenamine-Searle, 0.3 Gm. Ampules.—Each ampule contains sulpharsphenamine-Searle (THE JOURNAL, April 20, 1929, p. 1349) 0.3 Gm. G. D. Searle & Co., Chicago (Jour. A. M. A., June 15, 1929, p. 2021.)

Ampules Luminal-Sodium (Powder), 2 grains.—Each ampule contains 2 grains of luminal-sodium (New and Non-official Remedies, 1929, p. 81). (Jour. A. M. A., June 22, 1929, p. 2101.)

A NATIONAL CAMP-SAFETY CODE

The formulation of minimum standards of camp safety was decided upon at a recent meeting of representatives of 14 national organizations interested in summer camps. It was suggested that the research and secretarial work for the compilation of this "code" could be handled through establishing a graduate fellowship at Teachers College, Columbia University. For this purpose the National Bureau of Casualty and Surety Underwriters has offered a $1,000 fellowship for the coming year, open to candidates for a doctor's degree with comprehensive campaign experience.

—Children's Bureau.

BOOK REVIEWS

Books listed here become the property of the Ramsey and Hennepin County Medical libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

THE CONQUEST OF CANCER BY RADIUM AND OTHER METHODS. Daniel Thomas Quigley, M.D., F.A.C.S., Instructor in Surgery, University of Nebraska School of Medicine, Director of Radium Hospital, Omaha. 539 pages. Illus. Cloth, $6.00. Philadelphia: F. A. Davis Company, 1929.

GONORRHEA AND KINDRED AFFECTIONS. George Robertson Livermore, M.D., F.A.C.S., Prof. of Urology, University of Tennessee, and Edward Armin Schumann, A.B., M.D., F.A.C.S., Assoc. Prof. of Obstetrics, University of Pennsylvania. 257 pages. Illus. Cloth, $5.00. New York: D. Appleton & Co., 1929.

REPORT ON FOURTH INTERNATIONAL CONGRESS OF MILITARY MEDICINE AND PHARMACY. (Warsaw, Poland, May-June, 1927.) Commander William Seaman Bainbridge, M.C-F., United States Naval Reserve, Member of Permanent Committee, Delegate from the United States. 248 pages. Illus. No price listed. Menasha, Wisconsin: The Collegiate Press, George Banta Publishing Company.

CLINICAL LABORATORY METHODS (3rd edition). Russell L. Haden, M.A., M.D., Professor of Experimental Medicine, University of Kansas School of Medicine. 317 pages. Illus. Cloth, $5.00. St. Louis: C. V. Mosby Company, 1929.

OSTEOMYELITIS AND COMPOUND FRACTURES. H. Winnett Orr, M.D., F.A.C.S., Chief Surgeon Nebraska Orthopedic Hospital, Orthopedic Surgeon, Lincoln General Hospital. 208 pages. Illus. Cloth, $5.00. St. Louis: C. V. Mosby Company, 1929.

PRINCIPLES AND PRACTICE OF ELECTROCARDIOGRAPHY. Carl J. Wiggers, M.D., Professor of Physiology, School of Medicine, Western Reserve University, Cleveland, Ohio. 226 pages. Illus. Cloth, $7.50. St. Louis: C. V. Mosby Company, 1929.

THE TREATMENT OF FRACTURES. Lorenz Boehler, Chief Surgeon and Director Vienna Accident Hospital. Translated by M. E. Steinberg, M.S., M.D., Portland, Oregon. 185 pages. 234 illus. Cloth, $5.00. Vienna: Wilhelm Maudrich (Spitlagasse 1B, Ecke Mariannengasse), 1929.

ANNUAL REPRINT OF THE REPORTS OF THE COUNCIL ON PHARMACY AND CHEMISTRY OF THE AMERICAN MEDICAL ASSOCIATION FOR 1928. Cloth. Price, postpaid, $1.00. Pp. 75. Chicago: American Medical Association, 1929.

This book is a great deal more than a mere record of the negative actions of the Council on Pharmacy and Chemistry. It gives in full the reasons for the Council's rejection of various preparations, but it also records

results of the Council's investigations of new medical agents not yet out of the experimental stage, and frequently contains reports on general questions concerned with the advance of rational drug therapy. All three categories of reports are represented in the present volume.

Among the reports on products that have been denied admission to New and Non-official Remedies are those on Sanarthrit and Telatuten, two preparations of animal tissue, of indefinite composition, proposed for use in arthritis and arteriosclerosis respectively; on Clauden, a combination of lipoids and undefined proteins, proposed for use as a hemostatic; on Hart's Alimentary Elixir of Beef, a liquid medicinal food, "fortified" with glycerophosphates; on Alucol, claimed to be colloidal aluminum hydroxide and marketed under this nondescriptive name; on Oxo-Ate and Oxo-Ate B, claimed to be the ammonium and calcium salts, respectively, of orthoiodoxybenzoic acid and marketed under these proprietary, nondescriptive names; on Terpezone, stated to be pinene ozonide and marketed with exaggerated and unwarranted claims; on Vitalipon, an unscientific and indefinite mixture of lipoids claimed to be extracted from "vegetable and animal embryonic organs"; on Kalak Water, a solution containing sodium bicarbonate with many other ingredients of questionable utility, marketed under a nondescriptive name with unwarranted therapeutic claims; on Eu-Med, Aerosan Tablets, and Thyangol Pastilles, three shot-gun mixtures from Germany.

Among the preliminary reports are those on Metrazol, which has now been admitted to New and Non-official Remedies; on Phenylaminoethanol sulphate, a newly synthesized ephedrine substitute; on Ovarial-hormon Folliculin Menformon, the ovarian preparation originated by Dr. Laqueur of Amsterdam; and on Heparmone, a liver preparation.

The special report dealing with dextrose solutions containing cresol and intended for intravenous administration is a noteworthy example of the third category of Council reports we have mentioned.

WANTED—Salaried appointments for Class A Physicians in all branches of the medical profession. Let us put you in touch with the best man for your opening. Our nation-wide connections enable us to give superior service. Aznoe's National Physicians' Exchange, 30 North Michigan Ave., Chicago. Established 1896. Member The Chicago Association of Commerce.

BETHANY HOSPITAL, 3701 Bryant Avenue South, Minneapolis, is equipped to care for limited number of maternity cases at a nominal fee. New building. Light, airy rooms. Good food. Call Colfax 0016 or in person at above address.

WANTED—Young doctor to locate in established office, share reception room with dentist. Dentist has large practice. Fine opportunity for an up and going doctor. Write C. C. Gerber, Arcade and Case Streets, Saint Paul, Minn., or call Tower 7340.

DOCTOR'S AND DENTIST'S OFFICE FOR RENT —Established business and good location above drug store. Rent free to start. Address H. Harris, corner Thomas and Hamline, Saint Paul, Minn.

FOR SALE—Cheap. Scheidel Western Rectifying Motor. Used very little. Address D-30, care MINNESOTA MEDICINE.

WANTED—Position as anesthetist. Have completed six months' course in anesthesia at University of Minnesota, including some practice teaching. Address D-31, care MINNESOTA MEDICINE.

DESIRABLE LOCATION for young physician in growing district. 5161 Bloomington Avenue South, Minneapolis. Telephone Dupont 7964.

TECHNICIAN qualified to do *x*-ray and clinical laboratory work wishes position. Willing to start at reasonable salary. Address Esther M. DeSchene, 313 Park Street Anoka. Minn.

WANTED—Position as office assistant to physician and surgeon. Have nursing experience, also clerical and stenographic training. Can do physiotherapy and general laboratory work. Address D-32, care MINNESOTA MEDICINE.

X-RAY AND LABORATORY TECHNICIAN desires position in Twin Cities. Training received in Minneapolis hospital. References. Address D-33, care MINNESOTA MEDICINE.

FOR SALE—McCaskey desk. Two hundred open accounts above covered with roll top. One thousand accounts in drawers. Accessory filing cabinet four drawers. Complete with fillers and index. Light golden oak. New. Must sell. Half price. Address D-34, care MINNESOTA MEDICINE.